SHOUT
SHARE
IT

59.06(73)Xa
c2H

FOR THE PEOPLE
FOR EDVCATION
FOR SCIENCE

LIBRARY
OF
THE AMERICAN MUSEUM
OF
NATURAL HISTORY

Bound at
A.M.N.H.
1916

59.06(73)Xa

PROCEEDINGS

OF THE

SOCIETY FOR

EXPERIMENTAL BIOLOGY AND MEDICINE

VOLUME V

1907–1908

EDITED BY THE SECRETARY

NEW YORK

JULY 1, 1908

16-71815-Dec.1

PRESS OF
THE NEW ERA PRINTING COMPANY
LANCASTER, PA.

PREFACE.

In conformity with the custom inaugurated three years ago, a brief biography of the third president of the society is presented in this volume.

The *front* covers of the five numbers comprising this volume may be bound in the volume, if it is desired to mark off conveniently the communications of each meeting and to provide separate tables of contents. The printed matter on the rear covers of the numbers is given in the abstract of the executive proceedings (page 149) or in the list printed on page 157.

The numerals in parenthesis above the titles of the abstracts (pages 1–134) indicate numerical positions in the entire series of communications presented before the Society since its organization in 1903. The numerals in the index at the end of this volume correspond with those in parenthesis above the titles of the abstracts. Consequently none of the numerals in the index of this volume duplicates any of the numerals in the indices of the first four volumes. Convenience in reference was sought by the adoption of this plan of enumeration.

The recapitulation of the names of the authors and of the titles of the communications, presented on page 139, serves as an "author index." Only the numerals in parenthesis above the abstracts are indicated in it.

The constitution and by-laws have not been amended since their publication in Vol. IV.

NEW YORK,
July 1, 1908.

CONTENTS.

	Page.
Biography of Simon Flexner,	
Third president (1906–'08) of the Society for Experimental Biology and Medicine	v
Scientific Proceedings (25th–29th meetings):	
Communications of the twenty fifth meeting, October 16, 1907	1
Communications of the twenty sixth meeting, December 18, 1907	27
Communications of the twenty seventh meeting, February 19, 1908	55
Communications of the twenty eighth meeting, April 15, 1908	71
Communications of the twenty ninth meeting, May 20, 1908	101
Recapitulation of the names of the authors and of the titles of the communications	139
Classified list of contributing laboratories	147
Executive proceedings (25th–29th meetings)	149
Register of names and addresses of the members	151
List of officers	156
Classified list of members	157
Index of the scientific proceedings	159

Simon Flexner

SIMON FLEXNER.

THIRD PRESIDENT (1906–'08) OF THE SOCIETY FOR EXPERIMENTAL BIOLOGY AND MEDICINE.[1]

Simon Flexner was born in Louisville, Ky., March 25, 1863. At the age of nineteen he graduated in pharmacy and seven years later received the degree of Doctor of Medicine from the University of Louisville. His interest was attracted to the microscope at a time when in this country it was little used for medical instruction and during the second year of his medical study he inaugurated a course in histology, which he continued to conduct until after his graduation. In 1890 he pursued post-graduate medical studies at the Johns Hopkins University, giving special attention to pathology under Professor William H. Welch.

In 1890 Dr. Flexner was appointed Fellow in Pathology in the Johns Hopkins University. He subsequently held various positions in the pathological department of that University, being Associate Professor of Pathology from 1895 to 1899. In 1896 he studied in Strassburg and Prague. During this period he published observations upon a variety of subjects in pathological anatomy, general pathology and bacteriology, among which his studies of terminal infections and pancreatic fat-necrosis are especially noteworthy.

Early investigation of the relation of bacteria to disease had demonstrated the fact, now well known, that toxic substances, perhaps of albuminous nature, were the means by which micro-organisms caused injury. To a study of such substances and their mode of action, Dr. Flexner has given much attention; these investigations, begun by an examination of the histological changes caused by the toxins of diphtheria, have been continued in his study of the closely related vegetable poisons, ricin and abrin. His monograph on the Pathology of Toxalbumin Intoxication was published in 1897. An investigation of the cytotoxic substances produced by immunization of animals to various cells of a foreign species has stimulated much similar research in this country. Further studies have shown that the venoms of snakes resemble,

[1] Similar brief biographies of former presidents appeared in Volumes II and III.

in constitution, bacterial toxins and hemolytic sera, and have explained many facts concerning the mode of action of these complex substances.

Shortly after the close of the war with Spain, Dr. Flexner was sent to the Philippine Islands by the Johns Hopkins University as a member of a commission to study the diseases prevalent in these islands. Especial attention was given by him to tropical dysentery and its relation to the microörganism discovered by Shiga. Studies by Dr. Flexner and his pupils, continued after his return to America, have shown that the Shiga bacillus in one or the other of its types is associated with dysentery of this country and also with infantile diarrhea. Dr. Flexner was subsequently a member of the commission appointed by the National Government to determine if plague existed in San Francisco and to suggest means for its eradication.

In 1904 and 1905 an epidemic of cerebro-spinal meningitis occurred in New York City and the Health Department of the city appointed a commission to investigate the disease. As a member of the commission, Dr. Flexner undertook a comprehensive study of the meningococcus of Weichselbaum, the methods by which immunity might be produced in animals and the possibilities of serum therapy. This work, begun with a consideration of technical details with obvious interest only for the bacteriologist, has had far-reaching influence upon medical practice. It has pointed the way to the preparation of a curative serum which has reduced the mortality of cerebro-spinal meningitis.

In 1900 Dr. Flexner was called to the Professorship of Pathology in the University of Pennsylvania where he remained during four years. He was appointed to the directorship of the laboratories of the Rockefeller Institute for Medical Research in 1904 and after a year spent in the study of similar institutions in Europe, directed the establishment of the laboratories located in New York City. Dr. Flexner is a member of the National Academy, of the Philosophical Society and of the Association of American Physicians, a director of the Russell Sage Institute of Pathology and a member of the Advisory Board of the Hygienic Laboratory of the United States Public Health and Marine Hospital Service.

Dr. Flexner was a Vice-President of the American Association for the Advancement of Science, and Chairman of its Section of Physiology and Experimental Medicine, for the year 1906–'07.

SCIENTIFIC PROCEEDINGS.

ABSTRACTS OF THE COMMUNICATIONS.

Twenty fifth meeting.

College of Physicians and Surgeons, Columbia University. October 16, 1907. President Flexner in the chair.

1 (257)

Cardiac insufficiency due to high arterial pressure.

By **HAVEN EMERSON.**

[*From the Physiological Laboratory of Columbia University, at the College of Physicians and Surgeons.*]

Prof. Leo Loeb, of the University of Pennsylvania, first called my attention to the method of teaching the phenomena of pulmonary edema by administering massive doses of adrenalin to rabbits. His observations I repeated with similar, although not such severe results, in June, 1906, in one instance following the signs of mitral regurgitation and acute dilation of the heart through to a gradual recovery, and finally complete reestablishment of the normal heart sounds and heart rate in a rabbit.

During the winter of 1906–7 in the course of demonstrating the effects of direct and reflex vaso-motor phenomena to students, I had occasion to observe the effect of massive doses of adrenalin in cats, in which the carotid blood pressure and the intrapleural pressure variations were being recorded on a kymograph. The familiar phenomena of blood pressure, raised and maintained to 250–300 mm. of mercury, with a rapid respiration were observed, the usual slowing of the heart not appearing in these extreme conditions. In every instance there appeared shortly, small and then large moist rales over the entire chest, which gradually increased until the tracheotomy tube became flooded with serous froth. Often the systolic regurgitant murmur and the irregular rapid heart sounds were noticed preceding the edema.

If it had not been for a further observation I should not have ventured to bring this to your attention, but during June, 1907, I had occasion again to demonstrate the physical signs and physiological records in cats under heavy doses of adrenalin. To provide a more complete picture of the cardiac and pulmonary changes I used a cat under ether with the anterior chest wall removed and artificial respiration established. This cat, like another on an adjoining table, under normal respiration, showed the high blood pressure, and presently the moisture accumulating in the trachea. The heart in the cat with the chest opened presented the dilated right side of the heart, its deep venous hue and regurgitant murmur as usual, but presently these signs improved and the lungs from appearing deeply congested and sodden, became clear and pink, the serous exudate no longer obstructed the trachea, and the heart resumed its normal size, color, and rate. In the meantime the condition in the other cat was one of progressing edema.

In spite of the fact that cats, as well as rabbits, vary in their susceptibility to commercial adrenalin solutions, the changes in the picture above described, appeared too definite and too prompt to be due to any individual recuperative power or insusceptibility. I believe the change in the cat with the chest opened was due to the conditions of artificial respiration. Air was being forced into the lungs under pressure to distend them against their inherent elasticity and atmospheric pressure. Thus the pulmonary vessels may be considered as being subject to distinct positive pressure from without, recurring rhythmically with artificial respiration. It seems to me conceivable that this may have assisted the passage of blood from the right to the left heart, and in this way supplemented the right ventricle.

The effect of adrenalin in causing edema is not necessarily to be concluded a sufficient reason for avoiding its use therapeutically for relief from edema, since edema which has its origin in vascular relaxation, the vaso-paresis which occurs in the toxemia of pneumonia and diphtheria infections, as shown by Romburg and Pässler, is an entirely different clinical picture from the edema resulting from contracted arteries and failing heart, such as occurs in nephritics and severe arteriosclerotics.

In one instance adrenalin will improve the circulation by sub-

stituting its constricting action upon the blood vessels for the lack of vascular tone, and, as has been often observed clinically, will materially improve the existing pulmonary edema, while in the second instance, it would but aggravate the condition.

Although my observations are too few to base any general conclusions upon, I think they suggest that artificial respiration may be profitably tried in some cases of right heart failure, due to increased peripheral resistance, as soon as the mechanics of administering air can be perfected so as to avoid intubation or tracheotomy. And in this regard I may say that I have found that a person who is conscious can readily be supplied with air by artificial respiration by the use of an ordinary ether inhalation mask of the Bennett or Clover type. Whether this method will prove possible, in unconscious states or where there is general relaxation, as under anesthesia, I cannot say, but I think it is quite possible.

2 (258)

Effect of potassium cyanide upon metabolism.

By **GEORGE B. WALLACE** and **A. N. RICHARDS.**

[*From the Laboratory of Pharmacology of Columbia University, at the College of Physicians and Surgeons, New York.*]

Potassium cyanide was administered to two dogs in amounts sufficient to produce severe poisoning. One animal was kept upon a uniform, analyzed diet throughout the experiment (9 days) and the other was given no food during the experiment (5 days). In both animals an increase in the total nitrogen of the urine occurred on the day of poisoning which continued during the next following day. This increase was caused mainly by an increase in the amount of urea and by slight increase in the amounts of ammonia, total creatinin (sum of creatin and creatinin) and undetermined nitrogen (purin bases, uric acid, allantoin, amino-acids etc.). Neither experiment gave indication of marked percentage increase either of ammonia or of undetermined nitrogen, for the distribution of the nitrogen remained practically normal. Well marked absolute and percentage increase of creatin, and decrease of creatinin, was observed. Creatin was eliminated by the fasting dog throughout the whole period of observation.

Increase in the total sulphur eliminated was marked on the day of poisoning but, unlike the total nitrogen, returned to normal on the following day. The neutral sulphur was increased both absolutely and relatively to total sulphur on the poison day and that following. A corresponding decrease in oxidized sulphur was observed.

3 (259)

Pneumothorax and posture.

By **CHARLES A. ELSBERG.**

[*From the Department of Physiology and Pharmacology of the Rockefeller Institute for Medical Research.*]

The sudden entrance of air into the normal healthy pleura often gives rise to a train of grave symptoms. These symptoms have been studied experimentally in dogs. Most of the animals died from the pneumothorax when a large opening was made. Gluck lost all of the dogs he used; Biondi lost 4 of 5; Block, Marcus, Schmidt, Pourrat and Rodet, Tuffier, Murphy, Quenu and Longet, had similar experiences.

In some recent experimental investigations of open pneumothorax I obtained similar results. Many of the dogs either died suddenly as soon as an opening into the pleura was made, or a violent expiratory dyspnea ensued, soon followed by rupture of the mediastinal septum, double pneumothorax, and death. The method of operating on the dogs was the following: The animal was given a hypodermic injection of morphin, and a few hours later was anesthetized with ether; tracheotomy was performed, a cannula inserted, and the administration of ether continued through the cannula. One or more ribs were resected, the intercostal muscles divided, the pleura exposed, and a small opening carefully made in it and gradually enlarged. In a number of the animals, the pressure of the inspired and expired air was roughly measured by allowing the animal to breathe into a large bottle, the tube leading to it from the tracheal cannula being connected by means of a T-tube with a water manometer.

If a small opening (1–5 mm.) is made in the right or left pleura of a dog which is lying on its back, or on its right or left side, the

animal will in most instances continue to breathe well, although the amount of inspired and expired air will be less than (about two thirds of) the normal. That such an animal is very sensitive to the slightest influence which disturbs the breathing is shown by the fact that if the animal is deeply under the anesthetic, clamping the trachea for part of a minute will bring on the dyspnea; if the dog is not deeply under the anesthetic, the struggling of the animal or irritation of the cornea, mucous membrane, etc., will bring it on.

While most dogs stand a small opening well, sudden heart stoppage or dyspnea and death will follow in some instances. The larger the opening, however, the more likely is an occurrence of serious interference with respiration. In almost all of the animals in which the size of the opening approached to or exceeded that of the diameter of the trachea, dyspnea and death followed. No matter how slowly and cautiously the opening was made, typical expiratory dyspnea ensued, the heart becomes irregular and weak, the mediastinal septum bulged into the opening with each violent expiration, and rupture of the septum and double pneumothorax or sudden stoppage of the heart occurred. Nor did it make any difference in what part of the chest the opening was made, whether on the right or left side, near the apex or base of the lung, near the sternum or vertebral column. When, however, the animal was operated upon while lying flat on its belly, very different and gratifying results were obtained. When the dog is lying on his belly, not only can a small opening be made and the dog continue to breathe like a normal animal, but even a very large opening (2–4 cm.) can be made, and breathing go on regularly and quietly, almost if not quite like the normal. Even one half of one chest wall can be removed and the animal survive. Furthermore, if a dog on its back, with an opening in its chest 1 cm. in diameter and with typical violent dyspnea due to the pneumothorax, is turned on its belly, the breathing will often become regular and quiet again, and the pressure of air breathed in and out will be found to be several times as great as when the animal was on its back. On the other hand, a dog on its belly with a large opening in one pleural cavity and breathing quietly, can be brought into a condition of grave dyspnea and asphyxia by turn-

ing it on its back. Sometimes it is even possible to resuscitate an animal that has stopped breathing by thus turning it on its belly.

A dog on its back will sometimes stand a double pneumothorax when the opening in each pleura is a very small one, not more than 1–2 mm. in diameter and very slowly made. But with the dog on its belly, an opening almost 1 cm. in diameter can sometimes be made in each pleura, if cautiously done, and the animal will often continue to breathe and survive for hours. The amount of inspired and expired air in such an animal is surely small as compared with the normal, but it is apparently sufficient to keep the animal alive.

The explanation for this great difference between an animal on its belly and on its back with an open pneumothorax, is not an easy one. The following considerations and experiments are offered as a preliminary contribution to a solution of the problem.

The two pleural cavities are separated by the layers of the anterior and posterior mediastinal septa. Between the two lies the heart. In the dog, the posterior seems to be somewhat tougher than the anterior, and somewhat more fixed and tense. With violent respiratory movements, it is the anterior septum which more especially flaps to and fro and bulges when an opening in the pleura has been made, and it is the anterior septum which is so apt to rupture and thus cause double pneumothorax and death of the animal. When the dog is on its back, the heart falls backward and the bulging of the anterior mediastinal septum is made more easy. It is different when the animal is on its belly. The heart then falls toward the anterior chest wall and thus supports the anterior septum; hence the flapping of the septum, the interference with the respiration of the lung on the sound side, the bulging on expiration on the open side, cannot so readily occur. The following experiments, which I have repeated several times, are, I think, of importance in this connection.

After several ribs had been resected and before the pleura was opened, with the dog *lying on his back*, the pericardium was attached to the anterior chest wall by a suture, so that the heart was pulled forward. A very large opening could now be made in the pleura, almost as large as when the animal was on its belly, without causing the occurrence of serious symptoms. The

moment, however, that the stitch was cut and the heart allowed to drop backward, the typical pneumothorax symptoms appeared. In a second dog, *lying on his belly*, the pericardium was attached to the posterior chest wall by a stitch before the pleura was widely opened. The heart could therefore not drop toward the anterior chest wall. When an opening was made in the pleura of this animal, the typical pneumothorax symptoms started. They ceased soon after the stitch was cut and the heart allowed to drop toward the sternum. In this way it was often possible to reverse conditions; to put a dog with an open pneumothorax on its belly into the condition of one on its back, and vice versa.

From the above experiments, the conclusion may be drawn that in dogs, at least, pneumothorax is better borne when the animal is on its belly than when in any other posture. The reason therefore is to be found in the change in the position of the heart in the thorax when the animal is on its belly.

I may be permitted to add that I have in Mt. Sinai Hospital and in private practice operated upon a number of patients with empyema, upon a patient with a bronchiectatic cavity, on one with a subphrenic and one with a liver abscess and had the patients lying flat on the abdomen during the operation. In all of the patients the pleura had to be opened. The last three patients in whom an almost normal pleura was opened showed unusually few untoward symptoms when the opening was made and air entered the pleural cavity. In the patients in whom an operation for empyema was done, it was noted that with only one exception the coughing and interference with breathing that is regularly observed when the opening in the pleura is made for this affection, was entirely absent. Of course I am aware that these cases are too few to be conclusive.

At the French Surgical Congress of this year, Depage advised that all operations on the chest should be done with the patient lying on his abdomen; Kocher, in the last edition of his Operative Surgery, which has just appeared, states that in operating on the lung the patient should be on his back, and he adds in parenthesis "possibly also on the abdomen."

The writer desires to thank Dr. S. J. Meltzer, in whose department the work was done, for much aid and many suggestions.

4 (260)

The hypersensitiveness of the guinea pig to horse serum.

By **PAUL A. LEWIS.**

[*From the Antitoxin Laboratory of the Massachusetts State Board of Health, Boston.*]

The facts in this communication are related to the "Theobald Smith Phenomenon" first described by Otto. Additional observations in regard to the reaction and its pathology have been reported by Rosenau and Anderson, Besredka and Steinhardt, and Gay and Southard.

If a normal guinea pig be treated with a dose of 1/100 c.c. of normal horse serum alone, or of diphtheria antitoxic horse serum mixed with a suitable amount of diphtheria toxin, it becomes, after an interval of ten or twenty days, very susceptible to further treatment with normal horse serum. For a guinea pig whose preliminary treatment has been a toxin-antitoxin mixture, after an interval of three weeks, 5 c.c. of serum subcutaneously administered will frequently kill and will always develop symptoms. Five c.c. intraperitoneally is an almost certainly fatal dose. One one-hundredth c.c. given directly into the circulation is probably a certainly fatal dose.

As a sensitizing treatment I have employed chiefly the toxin-antitoxin mixture. As a test treatment I have administered the serum subcutaneously as a rule, but have occasionally injected serum into the circulation by the intracardiac method. Two c.c. is a safe dose for a normal animal by the latter method.

The results of other observers show that diphtheria toxin in some way increases the sensitizing power of the single small dose of serum. With serum alone the same effect can be produced by injecting fractions of the total small dose over a period of several days. In order to show the influence of the toxin, the intraperitoneal, intracerebral, and intracirculatory methods of testing must be avoided as the more vigorous reaction obtained when they are used masks the result. By injecting 2 c.c. of serum, intracardiac, into animals sensitized by toxin-antitoxin mixtures I have obtained a definite reaction on the sixth day. Careful

scrutiny of the results obtained by the subcutaneous test seems to show that the maximum of reaction is developed about the end of the third or fourth week. The incubation period is thus not sharply limited. The reaction is probably a slowly disappearing one but it may persist for a very long time — twenty two months in one of my cases.

The hypersensitive mother guinea pig transmits her abnormal condition to her offspring. In our experience about 50 per cent. of such offspring reacted. Members of the same litter tested with the same dose on the same day may either die very quickly or show almost no reaction. The fact of transmission from mother to offspring is strong presumptive evidence for the view that the reaction depends on the development of a special antibody by the sensitizing dose which is effective at the second treatment.

Such an antibody can be demonstrated by the passive transfer of the blood or blood serum of hypersensitive guinea pigs to normal guinea pigs. I have performed this experiment repeatedly. Thus, choosing hypersensitive animals that have reached the full period of reaction, I bled and injected 15 c.c. of defibrinated blood or blood serum into the peritoneal cavity of a fresh guinea pig weighing about 230 gms. After twenty four hours 1 or 2 c.c. of normal horse serum injected intracardiac killed this animal, which exhibited the typical symptoms. This fatal reaction at twenty four hours I believe to be distinct from the reaction obtained by Gay and Southard two weeks after the transfer of from 0.1 c.c. to 1 c.c. of hypersensitive blood. This latter reaction is probably due, as they supposed, to a retained horse-serum element or "rest" which actively sensitizes the animal. The hypersensitive antibody is not destroyed by heating to 60° C. for half an hour. Thus far further study of its properties has not been made.

If the hypersensitive animal is treated with a considerable, but not fatal dose of horse serum it is for some time less or not at all hypersensitive. This refractory or immune state can be very rapidly developed. The conditions are best illustrated by the following experiment.

Three hypersensitive guinea pigs were bled. Mixed blood was tested on a normal animal and found capable of transferring

the hypersensitive condition. Nine days later the animals were all treated as follows:

Sept. 19—8 p. m., 0.5 c.c. normal horse serum subcutaneously.
Sept. 20—8 a. m., 2.0 " " " " "
Sept. 20—12 m., 5.0 " " " " "
Sept. 20—8 p. m., 5.0 " " " " intraperitoneally.

None of the animals showed any marked symptoms under this treatment.

September 21, 10 a. m., one of the three received 1.5 c.c. normal serum intracardiac. No symptoms.

The other two animals were bled and 15 c.c. of the mixed defibrinated blood were at once injected into the peritoneal cavity of a fresh normal guinea pig weighing 240 grams. After an interval of thirty hours, this pig received 1.75 c.c. normal horse serum intracardiac. No symptoms.

Thus a complete immunity to the reaction can be developed in about twenty four hours and at the same time the blood loses its power to transmit the reaction, presumably because the antibody has been exhausted. The animal is probably in the condition of a normal animal which has had a single large dose of horse serum. Preliminary experiments make it seem possible, however, that the return to the hypersensitive state may be accomplished in less than the normal incubation period. This would be in analogy with the acceleration of reaction noted in cases of serum disease in human beings.

If normal guinea pigs are treated repeatedly with very small doses of horse serum ($\frac{1}{2000}$ c.c. – $\frac{1}{1000}$ c.c.) or with repeated non-fatal toxin-antitoxin mixtures, or if hypersensitive animals are fed with horse serum, they may subsequently be found hypersensitive in the usual way. In a percentage of cases, however, they will react differently when treated with 5 c.c. of horse serum subcutaneously. Acute symptoms are slight or absent. But at twenty four hours the animal may be very sick and have a large subcutaneous edema. The edema may progress and death occur on the second or third day, or in less severe cases the animal may survive with extensive cutaneous and subcutaneous necrosis. Post mortem examination on the third and fourth day shows interesting features.

The lung lesions described by Gay and Southard in the acute cases are here very slight. The gastric necroses are often very extensive. There are frequently large hemorrhagic necroses in the spleen. The mesenteric lymph nodes show desquamation of the endothelial cells of the sinuses. There are some red blood corpuscles in the sinuses. The endothelial cells are in various stages of degeneration, show some evidence of having acted as phagocytes, and the red blood cells are agglutinated around them in rosette-like arrangement. (Demonstration.)

In the spleen bordering the necrotic areas there is very marked phagocytosis of red blood corpuscles by large mononuclear cells. As contrasted with other instances of phagocytosis of erythrocytes this case has the following features. Usually but one corpuscle is found within the phagocyte. The enclosed corpuscle at first swells and has an increased affinity for eosin. Then the color reaction fades out and the corpuscle remains as a shadow which becomes smaller. Pigment is not common within these phagocytic cells. This form of phagocytosis with intracellular lysis is not a peculiarity of the guinea pig. In other conditions in this animal one finds phagocytic cells filled with numbers of rather shrunken deeply bronzed erythrocytes or with pigment particles evidently of erythrocytic origin. Comparison of the two classes of cases indicates, either that intracellular blood destruction in cells of endothelial type may be accomplished in ways that are fundamentally distinct, or that the factors of dissolution of the erythrocyte and segregation of the pigment constituents are independent and may run at different rates.

Recurring to the picture in the mesenteric lymph nodes, it seems possible that the endothelial cells may be the source of origin of certain serum hemagglutinins. It is an obvious suggestion also that agglutination functionally considered may be a constant factor in phagocytosis, serving to bring the subject cell, in this instance the erythrocyte, in contact with the phagocyte, in this case the endothelial cell.

This sharp reaction of the subcutaneous tissues resulting frequently in necrosis is closely analogous to the reaction which Arthus obtained to repeated subcutaneous doses of horse serum in the rabbit. It seems probable that it is a protecting factor so far as the animal is concerned.

5 (261)

A sporozoan found in the peptic glands of the common mouse.

By **E. E. TYZZER.**

[*From the Laboratory of the Caroline Brewer Croft Fund Cancer Commission of Harvard University.*]

This organism occurs frequently in the gastric glands of tame mice, but has not yet been found in wild mice, of which only a small number have been examined. It is extracellular in all stages of its development thus far observed. It presents structural characteristics without which it would be impossible for it to develop on the surface of the secreting gland-epithelium, and also produces structural changes in the gastric mucosa so that it is to be considered a true parasite. It is evident, from the morphological study of the various forms present, that this parasite has an asexual and a sexual mode of reproduction. All forms during their growth possess a definite limiting membrane at one point of which is a knob-like projection which represents an organ of attachment, evidently analogous to the epimerite of the Gregarinida. This projection gives to the organism a somewhat flask-shaped form. Occasionally a delicate thread extends outward from this projection, but I have been unable to determine whether or not this belongs to the structure of the organism. One or more globules, which are stained either by Sudan III or by osmic acid, are found in each organism. In ordinary preparations these appear as vacuoles. The developmental stages are briefly outlined in the following description.

Cryptosporidium muris, spec. nov. (unclassified). Development extracellular. In form flask-shaped, either spheroidal or ellipsoidal. All forms, during the period of growth, possess a relatively thin limiting membrane, an organ of attachment (epimerite?), and each contains one or more globules of fat which during segmentation are to be found in the residual body.

Schizont, after division of its chromatin, segments into eight banana-shaped merozoites, each possessing a demonstrable nucleus with a single karyosome. The substance of the organism is nearly all utilized in the development of the merozoites. Residual mass small and contains fat globule.

Microgametocyte smaller than schizont (never exceeds 5 × 3.5 microns). Division of chromatin followed by formation of sixteen or more microgametes which usually develop at the surface of the organism farthest from the organ of attachment. The larger portion of microgametocyte is left as a rounded residual body in which there is usually a fat globule.

Macrogamete or sporont, characterized by iodophilic granules and by the development of a dense capsule. Process of maturation and fertilization not determined. The entire organism is transformed into a single ellipsoidal spore which measures 7 × 5 microns and contains four sporozoites. Sporozoites about ten microns in length, slender, fusiform, without demonstrable internal structure. They lie parallel one with another within the capsule of the spore, and are bent in U shape around the centrally situated residual material.

Habitat. — Gastric glands of *Mus musculus*.

Since no description of this organism has been found, the above name *Cryptosporidium muris* is offered. No evidence has been obtained indicating that this organism represents a portion of the life cycle of any of the hitherto described parasites of the mouse, and it seems probable that it passes through its entire developmental cycle in the gastric glands. All the mice in certain cages are infected with this parasite, and since the ripe spores are found in the feces it is possible that infection follows the ingestion of contaminated food.

The systematic position of the organism appears uncertain. In its possession of an organ of attachment and of iodophilic granules, it resembles the gregarines. In its morphology, in the lack of motion in the adult, and in sexual dimorphism it resembles the coccidia. If it is to be included in the latter group, it may possibly belong to the Family Asporocystidæ (Döflein).

6 (262)

The role of tonicity in human isohemagglutination.

By **FREDERICK P. GAY.**

[*From the Laboratory of the Danvers Insane Hospital, Hathorne, Mass.*]

As was shown by Landsteiner and others, human bloods fall into three rather definite groups as regards isohemagglutination. The serum of members of group I agglutinate the corpuscles of groups II and III, but the corpuscles of group I are not agglutinated by any foreign human serum; members of group II agglutinate group III and are agglutinated by them; serum of members of group III agglutinate corpuscles of group II. Individuals in groups II and III, respectively, do not interagglutinate. It was found that constant relative differences in tonicity were present between these isoagglutinating groups, as was determined by the varying resistance, to hypotonic salt solutions, by the corpuscles of members of the respective groups, and by testing the tonicity of the various corresponding sera. Thus the tonicity of bloods of group I is found uniformly higher than that of bloods of groups II and III; bloods belonging to group II are higher in tonicity than those of group III. Simple hypertonic solutions of $CaCl_2$, but more particularly solutions hypertonic both in respect to NaCl and $CaCl_2$, produce a cohesion of human blood after several hours, that suggests isohemagglutination.

It is evident that hypertonicity alone, as regards total molecular concentration of all the substances present in serum, cannot account entirely for human isohemagglutination; although group II sera would agglutinate group III corpuscles, group III sera could not agglutinate group II corpuscles, as is the case. Relative differences in concentration of one or several salts or colloids could account for all the phenomena, as a given blood might be of higher molecular concentration in respect, say, to $CaCl_2$, and yet in total concentration be inferior to another blood, which it agglutinates.

It may be shown that when a serum of group I agglutinates a member of group II, and another of group III, the agglutinating power is apparently specific for each, as may be proved not only

by ordinary absorption methods, but also by working with an "agglutinin" bound to the cells and then split off by heat to 50° C. (Landsteiner). Such a "bound agglutinin" is highly "specific"; that is, if it has been bound by cells of group II it agglutinates only members of group II and not members of group III. Such an apparent demonstration of multiple specific agglutinins does not, however, rule out simple tonicity as the sole cause of the isoagglutination, for it may be shown that when serum I has been absorbed by blood II, *the tonicity of the serum has changed to equal the tonicity of II.* Such an absorbed serum would naturally no longer agglutinate II, but would still agglutinate III, since serum II will agglutinate III.

There is strong evidence, then, for the belief that isoagglutination of human blood may be due simply to physico-chemical variations in molecular concentration, and may be independent of the presence of any hypothetical new chemical bodies (agglutinins).

7 (263)

Effects of calcium and magnesium salts upon the development of rigor mortis.

By **S. J. MELTZER** and **JOHN AUER.**

[*From the Department of Physiology and Pharmacology of the Rockefeller Institute for Medical Research.*]

The effects of calcium and magnesium salts were studied on rabbits, cats, guinea pigs, rats and frogs; most extensively, however, on rabbits. Effects were obtained by means of subcutaneous, intravenous or intra-arterial injections; the intravenous method being more extensively employed than the other methods. The infusions were given in solutions of M/1, M/2, M/4, or M/8 concentration. Of the calcium salts we used $CaCl_2$, $Ca(NO_3)_2$ and $Ca(C_2H_3O_2)_2$; of the magnesium salts, $MgSO_4$, $MgCl_2$, $Mg(NO_3)_2$ and $Mg(C_2H_3O_2)_2$.

The following are the chief results. Calcium salts hasten and magnesium salts retard the development of the rigor of skeletal muscles. After treatment with calcium salts the rigor may begin twenty minutes after death, and after magnesium treatment it may

not begin before the lapse of six or seven hours. In the rigor after injection of a calcium salt the extensors mostly prevail irrespective of the original position. After treatment with magnesium salts the flexors prevail or the animal remains in the original position. The degree of the final rigidity after injection of a magnesium salt is not less than after similar introduction of calcium. The release from rigor appears earlier after injection of a calcium salt than after similar treatment with a magnesium compound. The administration of curare does not retard the calcium effect but it increases moderately the delaying effect of magnesium. The strong accelerating effect of a developed strychnin tetanus is not interfered with by magnesium. But the tetanus of a strong dose of strychnin can be completely suppressed and then the delaying effect of magnesium remains unimpaired. Calcium salts also hasten the heart rigor and magnesium salts delay it.

8 (264)

Restraint and promotion of tumor growth.

By SIMON FLEXNER and JAMES W. JOBLING.

[*From the Rockefeller Institute for Medical Research.*]

At a previous meeting of this society, we reported upon the promoting influence of heated tumor emulsion upon tumor growth in rats.[1] This evening we wish to report briefly the results obtained upon the re-inoculation of rats having tumors or having recovered spontaneously from them as affected by the injection of the heated emulsion of tumor cells and other substances.

The rats were grouped into several series as follows:

(*a*) Rats with tumors undergoing spontaneous absorption.

(*b*) Rats from which tumors after a degree of growth had disappeared spontaneously.

(*c*) Rats which failed to develop tumors on primary inoculation.

At the time these experiments were carried on, the sarcoma was at maximum virulence and gave approximately one hundred per cent. of successful implantations. Of these a certain number later underwent retrogression, as always happens with this tumor.

[1] Flexner and Jobling: This journal, 1907, iv, p. 156.

Before the effects of tumor and other emulsions are described, it is desirable to give the results of a control series of observations.

Into 249 rats already having one tumor were implanted second tumor fragments; 59 per cent. of these rats developed a second growing tumor. It was subsequently found that at the time of the second implantations some of the tumors were already undergoing retrogression, and as the rats which have recovered spontaneously are more refractory than rats with growing tumors, this percentage of successful secondary transplantations should properly be stated still higher.

Into each of 70 rats which had recovered spontaneously from growing tumors, a second implantation of tumor particles was made; 17 per cent. of these were successful.

Into 201 rats which had been inoculated once unsuccessfully with the tumor while its virulence was below the maximum, second implantations of the virulent tumor were made; 49 per cent. were successful.

With this series is to be compared the next to be given.

A group of rats in which the tumors were being absorbed or were entirely stationary was divided into halves. One half was injected with heated emulsion and ten days later with tumor. The tumor grafts grew in 60 per cent. of these animals. The other half received the tumor but no emulsion; the grafts grew in 36 per cent. The control series gave 100 per cent.

Taking a second group of rats in which the growing tumor was later absorbed, the same experiment was carried out. The rats not receiving the heated emulsion developed tumors in 9 per cent., while those receiving the emulsion developed tumors in 30 per cent.

We now return to a group of rats which having been injected with the heated emulsion and successfully inoculated with tumor subsequently recovered. A second injection of heated emulsion was given to one part and none to another. At the expiration of the ten day period, new tumor grafts were implanted with the effect of producing 30 per cent. of tumors in both series. Here again the control rats gave 100 per cent. of tumors. Hence it appears that no such discrepancy in promoting effects arises from a second as from a first injection of the heated emulsion. But what is equally surprising is the high percentage of successful secondary

graftings in this group of animals as compared with the low percentage in those spontaneously recovering without the emulsion, namely 30 as compared with 9 per cent. If, however, this group be compared with the group in which after spontaneous recovery heated emulsion was injected for the first time and followed by new grafts, the percentage of successful re-inoculations is identical in both, namely 30 per cent.

It would be premature to attempt a discussion of this interesting and unexpected fact, since it seems to imply that by the injection of the heated emulsion a state of susceptibility to tumor implantations can be preserved while, at the same time, the originally implanted tumor has suffered spontaneous absorption.

The next experiment was devised to determine the effects to be obtained from rat serum. Four sera were employed: (*a*) From normal rats; (*b*) from rats which did not develop tumors after repeated inoculations (normally immune); (*c*) from rats in which the tumors disappeared spontaneously (artificially immune); and (*d*) from rats with growing tumors. Bouillon and horse serum were used as controls. All specific effects were absent, that is, the sera neither inhibited nor promoted the growth of the grafts, while the bouillon seemed to be somewhat, and the horse serum rather more, inhibitory.

Finally, heated and unheated emulsions of various organs, liver, spleen, kidney, muscle and testicle were tested. No promotion or inhibition of growth was noted. With this experiment was combined still another test with the heated and unheated tumor emulsion. All the animals, controls included, received grafts of the same tumor on the same day, that is, ten days after injection of the emulsions. The control rats and the rats injected with organic emulsions gave 80 per cent. of successful implantations. The rats which received unheated tumor emulsion showed 60 per cent. and those which received heated tumor emulsion showed 100 per cent. of growths.

9 (265)

Reestablishment of function in transplanted kidneys.

By **ALEXIS CARREL.**

[*From the Rockefeller Institute for Medical Research.*]

Both kidneys, the left suprarenal gland, their vessels, their nerves and ganglia, the corresponding segments of the aorta and vena cava, the peritoneum, the ureters and a part of the bladder were removed in one mass from a female cat and transplanted in another female cat whose normal kidneys had been extirpated. The circulation was reestablished through the new kidneys and the flap of bladder sutured to an opening in the bladder of the host.

After this operation, the animal remained in excellent condition. She walked and played like a normal young cat. She was drinking milk and eating a great deal of raw meat. She grew very fat and enjoyed good health. She urinated abundantly —from about 60 c.c. to 255 c.c. per 24 hours. The density of the urine was very changeable, generally from 1.015 to 1.035. Urea was abundant owing to the large amount of meat digested by the animal, and amounted to from 2.7 grams to 5.1 grams per 100 c.c. During the first few days, the urine was a little bloody. The amount of albumin found was 0.50 gram and 0.25 gram for 1000 c.c. on two different occasions. It disappeared from the urine eight days after the operation. On the thirteenth day, albumin was found again, and increased progressively from traces to 1.5 grams and more per liter. Progressive enlargement of both kidneys was observed. Thirty days after the operation, the animal became suddenly ill, and died on the thirty first day. *Macroscopical examination*: enlargement of both kidneys due apparently to a compression of the renal veins by a large organized clot infiltrating the subperitoneal and perivenous connective tissue between the kidneys. *Microscopical examination*: slight acute diffuse nephritis and dilation of the blood vessels.

10 (266)

A depressor reaction obtainable by traction on the carotid artery.

By **TORALD SOLLMANN** and **E. D. BROWN.**

[*From the Pharmacological Laboratory of Western Reserve University, Cleveland, Ohio.*]

By pulling on the carotid artery, a very marked fall of blood-pressure (often of 50 to 90 mm. Hg) and increase of respiration can be produced. Our attention was directed mainly to the fall of pressure, but most of the remarks below apply also to the respiratory phenomenon. If the carotid is divided, the phenomena can only be elicited by traction of the cephalic, but not of the cardiac end. It is therefore not due to kinkage of the aorta. It occurs after both carotids are tied, so that it is not due to cerebral anemia. It does not occur on traction of either end of the cervical vagus, depressor, or sympathetic, so that it is not produced by accidental stimulation of these nerves. It does occur after all of these nerves are divided on both sides (in dogs, cats and rabbits) so that these nerves are not concerned in the phenomena.

The following remarks apply to dogs in which both vagi were divided. Oncometric observations show that the volume of the abdominal organs decreases with the fall of blood-pressure, so that this fall cannot be vascular. In further support of this conclusion, it was found that the fall occurs after section of both splanchnics, and after clamping the aorta at the diaphragm. The fall must therefore be cardiac. The heart rate is commonly somewhat slowed, but this is neither constant nor pronounced. Myocardiographic tracings show that the fall of pressure is indeed due to weakening of the cardiac contractions. We have tried to trace out the path which is taken by this highly peculiar and novel reflex. By following the carotid upward, we found that the reaction could be obtained from the region of the common carotid containing the origin of the internal carotid, but from none of the other branches, or at least only to a very slight extent. It could also be elicited by electric stimulation of this region. The internal carotid at this point is surrounded by a rich plexus of nerve fibers.

In trying to separate these from the artery, we were confronted by the peculiar difficulty that the reaction often disappeared suddenly — a good reaction would be obtained by pulling a bit of tissue, but on tying and dividing this, traction on either end would become ineffectual. We have here apparently a very peculiar condition of localized shock. This did not occur to the same extent in all animals, and furthermore, the reaction often reappeared after an hour. In this way we were able to show that it is localized in the nerve plexus rather than in the artery (we thought that the traction was perhaps transmitted by the artery to some intracranial structure but this is evidently not the case). When the carotid is pulled this nerve plexus is stretched and thus stimulated. We are wholly ignorant of the peripheral connection of these afferent fibers, of the conditions under which they are stimulated in the intact body, and of the functions which they subserve. As to the efferent path, our evidence so far is mainly negative.

As already stated, none of the cervical nerves are concerned, and the splanchnics are also excluded. The fall occurs equally well when the annulus of Vieussens and the inferior (middle) cervical ganglion with all its branches are excised on both sides. It therefore does not take the path of the ordinary accelerator or augmentor fibers. It occurs even after the greater part of both stellate ganglia are excised; but then the effect is diminished. However, we cannot yet state positively whether this diminution is due to the elimination of a part of the afferent path, or merely to the general shock attending the excision of these ganglia. We must conclude, however, that the depressing impulse reaches the heart by none of the known cardiac nerves. We do not know whether it acts by inhibiting some unknown augmentor nerves, or by stimulating some unknown depressor nerves. We are actively engaged in further investigation of this problem.

Other points worthy of mention are the following: The maximum fall varies with different animals; in some there is no fall, or there may even be a rise, but, on the whole, fall is the rule, especially in vigorous animals. When traction is applied, there is a latent period of several seconds, then a pretty abrupt fall, then a slight recovery, and then the pressure runs at a constant low level, so long as the traction is maintained or for at least ten min-

utes. When the traction is removed, there is again a latent period of several seconds, and then the pressure assumes its original level. Up to a certain point, the extent of the fall varies with the force of the traction; in most animals the maximal fall is reached when a weight of about 200 gm. is suspended from the divided carotid artery. A larger weight causes dyspneic convulsive respiration and may raise the blood pressure, probably by vaso-constriction.

The reflex is fairly resistant to most forms of shock. It occurs after curare, atropin, suprarenal, strychynin and phenol, and during depressor, splanchnic, and accelerator stimulation. When moderate doses of nicotin are injected, it is temporarily abolished, but reappears in a few minutes, whilst the vagus ganglia are still paralysed. This phenomenon can be reproduced by repeating the nicotin treatment.

11 (267)

A modification of Teichmann's method for obtaining hemin crystals, with a demonstration of specimens.

By **JAMES P. ATKINSON** and **ARTHUR I. KENDALL.**

[*From the Chemical Laboratory, Department of Health, City of New York.*]

This modification consists in heating suspected blood in a tightly plugged or sealed tube. Heating is best carried out by immersing the tube in boiling water. The reagents are those usually employed, *i. e.*, sodium chloride and glacial acetic acid. Heating is continued for fifteen minutes, at the end of which time the tubes are removed from the water bath and allowed to cool slowly at room temperature. After cooling, the tubes are broken open and the liquid poured into a watch glass or small evaporation dish and concentrated over the water bath. When the volume of the liquid has thus been reduced to a few drops, it is poured on a glass slide and covered with a cover glass. If sodium chloride crystals appear under the microscope a drop of water will dissolve them and leave the observation of the hemin crystals unobstructed. The treatment with the hot glacial acetic acid in the closed tube completely decolorizes the material on which the stain occurs. Heating in the closed tube keeps the condition of temperature and

strength of reagents constant. This method appears to yield larger crystals than those obtained by the old method.

12 (268)

The influence of ether anesthesia on the excretion of nitrogen by dogs.

By **PHILIP B. HAWK.**

[*From the Laboratory of Physiological Chemistry of the Department of Medicine of the University of Pennsylvania.*]

A series of nine experiments were made upon four dogs, the experiments ranging in length from 7 to 17 days. The plan followed was to get the animals into a condition of nitrogen equilibrium, then to induce ether anesthesia continuously for periods varying from ½ hour to 4½ hours. The initial influence of the ether was to cause an increased excretion of nitrogen, the average daily percentage increase varying from 6.5 per cent. after 30 minutes anesthesia to 43.5 per cent. after 3 hours narcosis. In the latter case the animal had been anesthetized on each of the two preceding days and therefore this large increase may represent the cumulative effect of the anesthesia. In six of the nine experiments the average daily excretion of nitrogen continued above normal during the entire experimental time after anesthesia was induced, the ultimate average daily increase varying from 3.5 per cent. for a 5-day period following 2 hours anesthesia to 17.5 per cent. for a 14-day period after ether narcosis had been induced for periods of 1, 2 and 3 hours on 3 successive days. In the three experiments which showed a diminished excretion of nitrogen after anesthesia the normal output of nitrogen was decreased 1.5 per cent., 1.8 per cent., and 4.0 per cent. as the result of anesthesia periods of ½ hour, 4½ hours and 1 hour respectively.

In each experiment the anesthetic had a diuretic effect, the extent of the diuresis varying from an initial average daily increase of 5.7 per cent. in the volume of urine after ½ hour of anesthesia to an increase of 24.8 per cent. after an anesthesia period of 4½ hours. The diuretic effect continued until the close of the experiment in every instance except one, the average daily percentage

increase in the urine volume for the entire experimental time ranging from 3.1 per cent. for 7 days after 4½ hours anesthesia to 20.7 per cent. for 7 days after 2 hours anesthesia. In one experiment narcosis for 1 hour caused a diuresis which was persistent throughout the remaining 10 days of the experiment during which time the urine output was increased a total of 710 c.c. or 18.4 per cent.

Anesthesia caused the animals to lose body weight, the percentage loss during the 24 hours following anesthesia varying from 1 per cent. to 3 per cent. The loss of weight following the anesthesia of a dog for periods of 1, 2 and 3 hours on three successive days was 6.5 per cent.

In all except two experiments the reaction of the fractions of urine first voided after anesthesia was amphoteric, alkalinity generally predominating. In the two experiments noted the urine was acid both before and after anesthesia. The specific gravity of the first urine passed after ether narcosis generally ranged from 1024 to 1042 as compared with a normal specific gravity ranging from 1015 to 1019.

13 (269)

The relative value of antitoxin and other curative substances in antidiphtheric serum.

By **EDNA STEINHARDT** and **EDWIN J. BANZHAF**.

[*From the Research Laboratory of the Department of Health, New York City.*]

Cruveilhier, in a paper in the *Annales de l'Institut Pasteur* (XVIII, p. 249), quotes Roux, Marfan, Martin and Momont as finding that the dose of antidiphtheric serum which is most efficacious is not always the one that contains the greatest number of antitoxic units. They assert that in the serum, there are, besides antitoxin, other substances whose therapeutic value is ignored in the present measurement of antitoxic units.

Cruveilhier carried out a series of experiments, in animals infected with diphtheria bacilli, and compared sera of different antitoxic strengths from several horses as to their value preventively and curatively. His results apparently showed that it was the

quantity of serum rather than the number of units of antitoxin which was efficacious. He drew the following conclusions:

The curative effect of the serum does not depend exclusively on the number of antitoxic units present.

The ordinary method of titrating antitoxin is not sufficient to determine the value of the serum.

We have investigated the same subject with results which do not agree with those of the above-named authors. In our experiments we infected the guinea pigs with three different strains of the diphtheria bacillus. We compared sera of the following unit contents: One of 200 units with one of 1000 units; one of 600 units with one of 1300 units; one of 600 units with one of 335 units, also with one of 200 units. The last three sera were obtained from the same horse during the course of immunization.

In both the preventive and curative experiments, we were unable to detect any protective substances which are not measured in the present method of standardizing antitoxin.

From the results obtained in the comparison of the antitoxic contents of sera we draw the following conclusions:

The therapeutic value of the serum depends upon the antitoxic content.

The present method of standardizing an antitoxic serum accurately measures its therapeutic value.

Twenty sixth meeting.

Rockefeller Institute for Medical Research. December 18, 1907.
President Flexner in the chair.

14 (270)

The effect of light on cells in fluorescent solution after addition of potassium cyanide.

By **ELIZABETH COOKE** and **LEO LOEB.**

[*From the Laboratory of Experimental Pathology of the University of Pennsylvania.*]

Since potassium cyanide inhibits certain oxidative processes in living matter such as respiration in plants and animals, the segmentation of fertilized egg cells, etc., behaving in this regard like an atmosphere of hydrogen, it might be expected that the destructive action of light upon cells suspended in fluorescent solutions, an action that is in part at least one of oxidation, would be diminished or obliterated by the addition of potassium cyanide to such solutions. On the contrary, the addition of very small quantities of potassium cyanide to sea water containing fluorescent substances (eosin, methylene blue, neutral red and combinations of these) causes a marked acceleration of the destructive action of visible light rays upon star-fish eggs immersed in these solutions. The chemical character of the fluorescent substances being different, it is evident that the chemical character of the fluorescent substance does not influence the action of potassium cyanide. But if star-fish eggs in such solutions be deprived of oxygen, by substituting for air an atmosphere of hydrogen, they no longer undergo deterioration under the action of light and this result remains uninfluenced by the addition of potassium cyanide to the solution. It asserts this accelerating action only if added to eggs in fluorescent solutions exposed to light. If the cells were first immersed in a solution of potassium cyanide in the dark and afterwards transferred to the fluorescent solution in the light, potassium cyanide did not show any effect. Also control experiments in which

small amounts of acid, alkali or certain alkaloids took the place of potassium cyanide showed no such results.

It would seem therefore that the oxidative processes, which perhaps in combination with other chemical processes, take place through the action of light upon cells in fluorescent solution, must be of a different character from the oxidative processes taking place in respiration and in cell division, since the former are accelerated instead of, like the latter, being inhibited by the action of potassium cyanide. Since in a hydrogen atmosphere the addition of potassium cyanide does not cause this specific effect, the action of potassium cyanide in markedly increasing the destructive action of light cannot be a primary one but can only follow or accompany the primary oxidation processes. In contradistinction to the oxidative processes of respiration these oxidative processes are not prevented or inhibited by potassium cyanide.

As to the way in which potassium cyanide produces this accelerating effect upon the action of light in fluorescent solution, there may be a number of explanations suggested. It is conceivable that the potassium cyanide accelerates certain oxidative processes in the protoplasm. Analogies for such an accelerating action exist in the behavior of potassium cyanide toward certain inorganic catalysers. According to Loevenhart and Kastle potassium cyanide accelerates the splitting action of copper or iron on hydrogen peroxide. On the other hand, potassium cyanide not only inhibits the action of ferments like catalase and oxidative ferments but also hydrolytic catalysers. Potassium cyanide might therefore in the cases which we are considering, exert an influence on hydrolytic processes which are perhaps secondary to the primary oxidations caused by light. But it is also conceivable that the potassium cyanide does not produce its effect by acting directly upon the cell protoplasm but that, through its addition to the fluorescent substance it brings about some change in the character of the radiant energy whereby the disorganizing oxidation is accelerated. Whichever of these explanations may prove to be the correct one it is certain that under the conditions of these experiments potassium cyanide does not prevent oxidative processes in the cells.

15 (271)

Physiological Age.

By C. WARD CRAMPTON.

[*From the Department of Physical Training, Board of Education, New York City.*]

The term *physiological age* refers to the stage of development in contradistinction to age in years and months, which is the usual method of designating age. Various evidences of physiological age are tooth appearance, pubescence, change of voice, menstruation, menopause, etc.

Pubescence is an evidence of sexual ripening and the beginning of adolescence. For the purpose of record, three physiological groups are distinguished corresponding to three successive stages of development (1) prepubescence, (2) pubescence, (3) postpubescence. The first group is characterized by an absence of hair upon the pubis, the second is an intermediate stage, the third group have hair upon the pubis. The data of the results below are taken from 4,500 New York High School boys, and are divided into half year age groups designated by the middle age value. The following table shows the percentage composition of each chronological age:

Age.	12.75	13.75	13.75	14.25	14.75	15.25	15.75	16.25	16.75	17.25	17.75
Prepubescent	69%	55	41	26	16	9	5	2	1	0	0
Pubescent	25%	26	28	28	24	20	10	4	4	2	0
Postpubescent	6%	18	31	46	60	70	85	93	95	98	100

It gives also the relative size of the subgroups. These facts have been hitherto disregarded and the chronological age group treated as if it were homogeneous.

These subgroups in each age exhibit characteristic differences in physical measurements which differences are far greater than the difference between contiguous year groups.

At the age of 15.75 the postpubescent group (85 per cent. of all) are 34 per cent. heavier, 32 per cent. stronger and 9 per cent. taller than the prepubescent group (forming 5 per cent. of all at the same age) as indicated on the next page:

	Weight. kilos.	Height. cm.	Strength. kilos.
Prepubescent.	36.7	149.8	32.5
Pubescent.	41.8	153.1	30.4
Postpubescent.	49.3	162.6	42.9

The differences in weight, height and strength between prepubescents and postpubescents of the same age are equal to the differences between age groups that are 6 to 8 years apart.

Conclusion. — Age groups are heterogeneous and cannot serve as a unit for reference and experiment. We must substitute groups based upon physiological age.

16 (272)

Gastric peristalsis after section of the vagi and splanchnic nerves.

By **JOHN AUER.**

[*From the Department of Physiology and Pharmacology of the Rockefeller Institute for Medical Research.*]

Some time ago I described a method by means of which gastric peristalsis in the rabbit could be studied under normal conditions, without any operative interference whatsoever. The method is briefly as follows: If the hair of the epigastrium of a rabbit, fed well two hours before, is cut short, the stomach is largely outlined through the abdominal wall, and the peristaltic waves may be studied by inspection or by registering the waves with a tambour.

As the stomach is provided with extrinsic nerves (vagi and splanchnic) and with an intrinsic system (plexuses of Meissner and Auerbach) it was of interest to study the mobility of the stomach when deprived partly or entirely of its extrinsic innervation. This question has been studied by a number of observers, most recently and thoroughly by Cannon who used the X-ray method on cats. By means of the simple and physiological method outlined above the effects of sectioning the vagi or the splanchnics or both were studied. The vagi were, in this series, invariably cut below the diaphragm in order to preserve the vagus innervation of the thoracic viscera.

First series. Subdiaphragmatic section of the vagi only.—Two

hours after operation the tambour registered only very slight volume changes of the stomach. Inspection showed no visible peristalsis. After some days peristalsis was again normal.

Reflex stoppage of peristalsis resulted from strong odors, sudden movements before the animal's eyes, loud noises, pain, just as in normal animals.

Second Series. Section of the splanchnic nerves only.—As a rule good, effective peristalsis occurred less than one hour after the operation. The waves, however, are slower, and the period of contraction seems to be of longer deviation than normally.

Regarding the reflex effect of sensory stimuli, the data are as yet too few to permit a definite statement, but it seems as if the ordinary stimuli employed exerted no marked effect on the course of stomach peristalsis.

Third Series. Section of vagi and splanchnics.—After section of all the extrinsic nerves peristalsis usually appeared within one hour but was not normal until some days after the operation.

The various sensory stimuli, which in the normal animal caused inhibition of the stomach movement, were now ineffective. The stomach, however, showed periods of inactivity alternating with periods of strong peristalsis. As these stoppages bore no definite relation in time to any stimulus applied to the animal, they were probably due to inhibitory influences exercised by the plexuses of Meissner and Auerbach.

These results confirm the statement that of the extrinsic nerves the vagi are chiefly motor and the splanchnics chiefly inhibitory for the motility of the stomach.

They also show that apparently normal stomach peristalsis is by no means dependent upon the extrinsic nerves, that the local mechanism is amply sufficient after some time.

The results show that the local nervous mechanism in the walls of the stomach must depend largely under normal conditions upon the directing influences of the vagi and splanchnics, for it usually takes days before the local government is able to produce strong, powerful, well-coordinated gastric waves.

17 (273)

The effect of stimulation of the vagus nerves upon the development of rigor mortis of the mammalian heart.

By **DON R. JOSEPH** and **S. J. MELTZER.**

[*From the Department of Physiology and Pharmacology of the Rockefeller Institute for Medical Research.*]

Stimulation of motor nerves hastens the onset of rigor mortis in the corresponding skeletal muscles. Cutting the nerves retards it, the retardation being due, it is believed, to the elimination of subminimal nerve impulses. Would the retardation of the rigor be still greater if inhibitory nerves could be stimulated? This question is not applicable to skeletal muscles, but it is a definite problem with reference to the onset of rigor in the heart muscle. Would a prolonged effective stimulation of the vagus nerves retard the onset of cardiac rigor? There were reasons to expect that the effect of such a stimulation would be indeed a retardation. The action of the cardiac vagus is inhibitory and the reverse of the action of a motor nerve; we might then expect that the effect of its stimulation upon cardiac rigor would also be the reverse of the effect of stimulation of a motor nerve upon the skeletal muscles, that is, retardation instead of hastening. Furthermore, increased muscular activity hastens the onset of rigor; it seemed reasonable to anticipate that the diminished activity, such as frequent standstill or slowing of the heart, would retard the onset of its rigor.

We have studied this question in 42 dogs, 16 cats and 10 rabbits. Both vagi were stimulated for half an hour before death and frequently also after death. Death was caused uniformly by bleeding and opening of the thorax. The outcome was a surprise; the obtained results were just the reverse of what was expected. But the results were uniform and unmistakable. We shall state them very briefly. They are as follows:

In all animals in which the vagi were stimulated, left and right ventricles stopped beating after death sooner than in the controls. The interval between the time of death and the beginning of the rigor in the left as well as in the right ventricle is in the experimented animals shorter than in the controls. The time elapsing

between the beginning of rigor and the attainment of its maximum is in the stimulated animals again shorter than in the controls.

We shall not burden our present statements with figures or other details. The essential point in our results is that with regard to the cardiac rigor, stimulation of the inhibitory nerves had the same effect as that obtained by stimulation of motor nerves upon skeletal muscles, although the two kinds of nerves have opposite functional characters. How is this puzzling result to be explained? We are inclined for the present to give our results the following interpretation. It is known that anemia and venous stasis hasten rigor. The onset of rigor in the lower extremities of a living animal following compression of the abdominal aorta is a well-known experiment. We believe that the frequent standstills and slowing of the heart with its attendant anemia, venous stasis and asphyxia of the tissues is the cause of the hastening of cardiac rigor in the animals whose vagi were stimulated. In support of this interpretation we may cite the fact that the rigor of the skeletal muscles also sets in earlier in the animals whose vagi were stimulated than in the controls, a fact for which the disturbance of the circulation seems to be the only possible explanation.

18 (274)

The antagonistic action of calcium upon the inhibitory effect of magnesium.

By **S. J. MELTZER** and **JOHN AUER.**

[*From the Department of Physiology and Pharmacology of the Rockefeller Institute for Medical Research.*]

Calcium and magnesium are chemically closely related elements. They are also close companions in the tissues of the animal body. It is the prevailing view that the physiological effect of both elements is similar in character. Many physiologists are at present of the opinion that calcium as well as magnesium exerts an inhibitory influence in the functions of the animal body. Loeb published in 1899 his observations of the inhibitory action of calcium upon the twitchings of frog muscles brought on by solutions of sodium chloride. It was then assumed by Loeb that all

the members of the group of alkali earths possess inhibitory properties including at first even barium. In the numerous subsequent papers by Loeb and his pupils the discussion turned, however, essentially around the inhibitory effect of calcium. Outside of the effect of calcium upon the heart, with reference to which Howell and his followers insist, contrary to the teachings of Loeb and his school, that calcium is an exciting and not an inhibiting agent, the opinion is now widely accepted that calcium is an inhibiting factor in the animal organism. It found its way also into pathology. For instance, a number of German and Italian writers hold the view that tetany of children is due to a diminution of the calcium content of the brain.

As to magnesium we have within the last few years published several studies in support of the hypothesis that magnesium salts favor inhibitory processes. The first fact which gave rise to that hypothesis was demonstrated in 1899 to the American Physiological Society when an intracerebral injection of a few drops of a solution of magnesium sulphate caused a state of paralysis in a rabbit while the injection of other solutions brought on convulsions.

In a series of recent studies which we have carried out upon the relations of the effects of calcium to magnesium, many remarkable facts came to light, all of which demonstrate unmistakably that calcium is the most available agent to neutralize the inhibitory effect of magnesium. We shall not enter here upon details ; we wish to show only the following striking and instructive experiment.

By subcutaneous injections of a magnesium salt rabbits are brought to a profound state of anesthesia and paralysis. The slow and shallow respirations indicate the approaching danger. Now 6 or 8 c.c. *m*/6 or *m*/8 solution of a calcium salt is given through the ear vein. Within a few seconds the respiration becomes quicker and deeper and within one minute the animal turns over, sits up and appears normal.

Here calcium not only did not add an inhibitory effect but completely neutralized the profound inhibitory effect of magnesium. The companionship of calcium and magnesium within the body means, at least in many instances, not a concerted action of similar effects but rather a resultant effect of antagonistic actions.

We may add that the experiment calls to mind similar relations

existing in plant physiology ; the retardation of growth on account of the presence of too much magnesium in the soil is promptly corrected by the addition of a calcium salt ; the process is termed "liming." In animals, therefore, as well as in plants calcium is antagonistic to magnesium.

19 (275)

Remote result of the transplantation of a segment of popliteal artery from a man to a bitch.

By **ALEXIS CARREL.**

[*From the Rockefeller Institute for Medical Research.*]

I have shown that a segment of carotid artery of a dog transplanted onto the aorta of a cat can act as artery for one year at least. In order to ascertain whether the same result is obtainable when the animals are zoologically more distinct than cat and dog, segments of human arteries have been transplanted in dogs. One of these animals was shown to the Society.

Seven months and twelve days ago, the abdominal aorta of the bitch shown to the society was severed, and a short segment of human popliteal artery was sutured to its cut ends. This popliteal artery belonged to a young man whose thigh was amputated by Dr. Ellsworth Eliot for an osteosarcoma. The vessel had been preserved in Locke's solution and kept in cold storage during the twenty four days which elapsed between the amputation and the transplantation.

After the transplantation, the bitch remained in excellent health and the pulse of the femoral arteries was normal. Five months and twelve days after the operation, an exploratory laparotomy was performed. The circulation of the aorta was found normal and the popliteal artery in about the same condition as at the time of the operation. Seven months and twelve days after the operation, the pulse of the femoral arteries is still normal.

This experiment shows that an artery, transplanted under certain conditions from man to dog, can act as artery for seven months at least.

20 (276)

Concerning the relation of the coagulation time of the blood to thrombosis in phlebitis.

By **HARLOW BROOKS** and **B. C. CROWELL.**

[*From the Pathological Laboratory of the New York University and Bellevue Hospital Medical College.*]

The etiological factors or conditions concerned in the production of thrombosis may be briefly summarized as:

(1) Those due to central or peripheral slowing of the blood stream.

(2) Those associated with lesions in the walls of the blood vessels.

(3) Alterations in the blood itself, such as tend to favor coagulation.

In actual cases, it appears without exception that two or more of these factors are associated in the production of thrombosis. Although the etiological agents mentioned above are generally accepted as the correct interpretation of thrombosis, it must be remembered that experimentally, as well as clinically, very discordant results are reported as to the causation of thrombosis, notably such as occurs in clinical phlebitis. Until more certain data are secured in regard to the process, but little can be expected in the way of successful prophylactic treatment or in the certain prognosis of those instances in which this lesion is to be feared.

The object of this brief study has been an attempt to show to what extent increased and decreased coagulability of the blood, artificially induced, may play a part in the production of thrombosis, or, expressed, in other words, whether in conditions productive of phlebitis, thrombosis is more likely to occur when the coagulation time of the blood has been lowered artificially or less apt to take place when analogous artificial means have been employed to prolong the coagulation time of the blood.

Our experiments have been conducted on a series of young and healthy rabbits, one third of which have had their coagulation time reduced by daily administration of 2 gms. of calcium lactate, an equal number of animals whose coagulation time has been arti-

ficially lengthened by daily dosage of 2 gms. of citric acid and one third used as control animals. The drugs were introduced into the stomach by tube feeding and it was found possible, with this dosage to reduce the coagulation time of the blood in the calcium lactate animals one half and to lengthen it in the citric acid animals about one third. Corroborating work already well authenticated, the maximum effect of these drugs takes place about two hours after their administration, and probably entirely passes away within twelve hours, especially with the rapidly excreted calcium salts. In our animals the drugs were continued in daily doses throughout the experiment, since it was found that effects were quite as apt to occur several days after the initial injury as immediately on its infliction, thus more closely approximating the conditions as they occur in man.

When autopsies were to be performed the animals were chloroformed and while the heart action was still vigorous a carotid artery was opened and the animal suspended so that the blood was very generally emptied from all the vessels of the body, and post-mortem clot or fibrin could not become confused with the true thrombi. We believe this to be an important step in the technic.

Our experiments may be grouped in two series: Series I comprises local injuries produced in and around the ear veins. Nine experiments of this character were performed. Series II includes attempts to produce vascular lesions predisposing to phlebitis and thrombosis by intravenous injections of various irritants. Five sets of experiments.

Series I; *Experiment a.* — A segment (3 cm.) of the distended marginal vein was isolated by compression between two artery clamps and the intervening vein distended with blood was intermittently compressed with toothed forceps for five minutes when minute hemorrhagic extravasations along the course of the trunk were demonstrable. The isolating clamps were then removed and the circulation allowed to become reëstablished. The resulting perivenous inflammation was slight and no thrombosis followed either in the calcium citric acid or control animals.

Experiment b. — Clamping the marginal vein with ordinary paper clips, cutting off at the time the anastomosing circulation, for thirty minutes, was followed by immediate reëstablishment of the normal blood flow in all animals without subsequent results.

Experiment c. — A 2 c.m. segment of the marginal vein was clamped and isolated with paper clips for twelve hours, the anastomosing circulation being meanwhile prevented. Immediately on the removal of the clips the circulation was reëstablished in all animals, the absence of thrombosis in these experiments being like the results of the experiments reported by Baumgarten and his student Rizor who was able to compress the vein for an even greater length of time without resulting thrombosis. After three days considerable inflammatory reaction developed about the site of some of the clamps, this being most marked in the calcium lactate animal, where finally a small segment of the vein became thrombosed. The inflammatory reaction was less marked in the control animal and there was still less reaction in the citric acid rabbit, thrombosis being absent in both. From these experiments one may conclude that mere stagnation of the venous blood produced no marked tendency toward thrombosis in the ear veins but that inflammatory lesions, with consequent phlebitis are more extensive in the case of the calcium animal and thrombosis may occur at the immediate point of injury of the vessel walls.

Experiment d. — A quantity of 24-hour growth of virulent pneumococci in bouillon was injected about the ear vein, the injection being continued to such a point as to cause compression anæmia of the desired segment of the vein. The circulation was shortly reëstablished and the amount of subsequent inflammatory reaction was slight. No thrombosis occurred in any of the animals. Our results in this experiment are therefore quite unlike those of Von Talke where, however, the coagulation of the blood was not altered, for Von Talke claims to have regularly produced thrombosis in this way.

Experiment e. — Five minims of 5 per cent. $AgNO_3$ were injected into the perivenous connective tissue of the ear. Immediate permanent thrombosis of the adjacent vessels followed. No difference in extent or degree existed between the three animals. This experiment was repeated using 1 per cent. solutions of $AgNO_3$. Slight perivascular inflammation without thrombosis resulted and was of about equal severity in all the animals.

Experiment f. — Three drops of pure turpentine was injected between the branches of the median ear vein. This was followed

within twelve hours by edema and an active inflammatory exudation with thrombosis of the involved vein in all three animals. The thrombosis was notably more extensive and resolution most delayed in the animal which had received the calcium lactate, less so in the control and least of all in the rabbit which had been poisoned with citric acid.

The same relations as regards severity and occurrences of lesions followed when 50 per cent. turpentine in inert oil was employed in the experiment except that the resulting inflammation and subsequent thrombosis was longer delayed.

Series II. *Experiment a.* — Fine comminuted sterile pumice was injected into the marginal ear vein in an attempt to see if the resulting thrombosis following probable embolism of the terminal arterioles would be more extensive in those animals which had received the calcium salt and less so in that which had received citric acid. All three animals recovered perfectly from the operation and later autopsies showed no lesion whatsoever which could be attributed to this injection.

Experiment b. — Fifteen minims of sterile cod liver oil was injected into the marginal ear veins of the three animals. None of them showed symptoms and later post mortems showed no lesions attributable to the experiment.

Experiment c. — Two minims of a pure 24 hour culture of virulent typhoid bacilli were introduced through the marginal vein. No symptoms of illness followed and these animals were subsequently utilized in experiment *d.* These results are exactly contrary to those of Jakowski who in similar experiments in guinea pigs and rabbits obtained almost constant thrombosis, but without the associated employment of calcium or citric acid.

Experiment d. — Fifteen minims of a suspension of a 36 hour broth culture of virulent typhoid bacilli in an equal bulk of sterile cod liver oil were introduced through the marginal vein. This was done in the expectation that embolism caused by the oil would be likely to afford sites for the growth of the bacilli and probable resulting thrombosis as described in the experiments of Jakowski. All the animals became seriously sick and on post mortem examination multiple serous petechiae, general parenchymatous degeneration, lymphadenitis and frequent infarctions

were found, but no general or isolated thrombosis. These lesions were notably most extensive in the calcium lactate animal and least so in the citric acid, but when the experiment was repeated under similar conditions, exactly opposite results followed, which leads us to believe that chance was really the controlling factor in determining these changes.

Positive results have been obtained in but a single set of experiments ; those were where terpentine was injected.

In so far as the results of this preliminary study go, one is led to the conclusion that thrombosis is most readily induced where active inflammatory lesions exist in the blood vessels probably associated in most instances with secondary degenerative changes. Purely mechanical lesions are much less apt to be productive of conditions favorable to thrombosis as a sequence of phlebitis.

Marked artificial increase or decrease in the coagulation time of the blood by the use of calcium lactate or citric acid, does not render animals abnormally prone to thrombosis incited by changes other than inflammatory.

Where true phlebitis exists, thrombosis is apt to be more extensive and less readily resolved, when the coagulation time of the blood has been shortened by the use of calcium lactate, and it is less extensive and more quickly absorbed where the coagulation time has been increased by the administration of citric acid.

Experiments as yet incomplete, appear to suggest that the rapidity or slowing of the general circulatory flow has but little bearing on the relative production of thrombosis in phlebitis, much less than clinical and anatomical observations have generally led us to think. We have also been led to suspect that the presence or absence of anastomoses of abundant degree is largely concerned as a factor in determining the location and extent of thrombosis in phlebitis.

21 (277)

The reactive power of the white rat to tissue implantation. Second communication.

By **ISAAC LEVIN.**

[*From the Department of Pathology of Columbia University, at the College of Physicians and Surgeons.*]

At the November meeting of the New York Pathological Society,[1] I reported briefly on a series of experiments I have undertaken on white rats, with the aim to investigate whether the great success attained lately in the transplantation of malignant growths from one rat into another is not due, in part at least, to the different behavior of the organism of the white rat to tissue implantation, as compared with other laboratory animals.

The majority of the workers who have experimented with the tumors of the white mouse or rat seem to be of the opinion that the success in the transplantations of these tumors is due to the great intrinsic power of limitless proliferation of the cells of these tumors, and they ignore the possible cellular reaction of the organism of the host to the implanted tissue. But the fact that pieces of the same tumor from the same rat or mouse grow readily on white mice of one race, and not at all on the white mice of another race; the fact, further, that while the original tumor, when implanted, grows in only about 10 per cent. of animals used, and, when re-implanted, it grows in 90 per cent., and some other similar instances, seem to indicate, *a priori*, that the white mouse or rat reacts differently to implantation of tumor, as well as of normal tissue, than the other laboratory animals.

My first set of experiments consisted in the implantation of normal tissue from one animal into another. Pieces of skin, liver, spleen, testicle, or mammary gland are implanted under the skin or in the peritoneal cavity of another animal. In the great majority of the experiments (about 60 to 75 per cent.) the pieces remained unabsorbed for as long as three weeks, even when, as in the peritoneum, they do not become attached anywhere, but float like a foreign body. Usually, the pieces are either surrounded by

[1] Levin: *Medical Record*, December 14, 1907.

a capsule consisting of an outer fibrous layer, and an inner one made up chiefly of round cells, or else the round cells invade the implanted tissue. One is impressed with the great amount of organization going on around the implanted pieces. On the other hand, the pieces themselves seem to retain the cell structure much longer than is usual in other animals.

It seemed interesting to investigate whether the white rat will also react differently from other animals to an introduction of an irritating foreign body.

An emulsion of aleuronat produces, in the ordinary laboratory animals, an abscess, when injected under the skin, and empyema, when injected into the serous cavity. In nine rats an *emulsion of aleuronat* was injected under the skin, and in each animal there developed in the place of injection a freely movable tumor the size of a small nut, which on examination proved to be a cyst filled with thick cheesy detritus. In only two animals did the skin become eroded; in the others the growth retained the same size during two to three weeks.

In six white rats I introduced into the peritoneal cavity one or two *triturate tablets of aleuronat.* The animals were killed in three days to two weeks after the operation. At the autopsy there was found in the place of each tablet a little growth of white grayish color, the consistency of a granulation, loosely adherent to the omentum, occasionally with some cheesy detritus in the center. Microscopically the growth consists of a conglomeration of round cells and leucocytes towards the center, and round cells and some beginning of fibrous connective tissue formation towards the periphery.

While even this last named formation is not a tumor in the true meaning of the word, but a connective tissue reaction to an irritation, it seems to be clear from all these experiments that the reactive cell proliferation and cellular organization is much more extensive in a white rat than in the other laboratory animals. On the other hand the absorption of the implanted tissue is a great deal slower, consequently there is a better chance for the implanted cell to proliferate and for the implanted tissue to grow. Whether there is still a qualitative difference between a tumor cell and a normal tissue cell, or conditions may be found under which normal cells will acquire the power of limitless proliferation, for the

study of this question the white rat is certainly the most appropriate animal.

22 (278)

The hemolytic reactions of the blood in dogs with transplantable lymphosarcoma.

By **RICHARD WEIL.**

[*From the Huntington Fund for Cancer Research, of the General Memorial Hospital, Loomis Laboratory, Cornell University Medical College, New York City.*]

Dogs with lympho-sarcoma in every stage of growth were made use of, including those in which growth was active and progressive, those in which the tumor was quiescent, those in which the growth was regressing, and those in which recovery with complete absorption of the tumor had taken place. Some of the dogs were in good general condition, others were cachectic. Controls were also made use of, including dogs in poor and in good condition. In all, the material comprised 34 dogs, 18 with a tumor history, and 16 without. These were all bled from the femoral artery or jugular vein. In all of these dogs, blood was obtained for serum, and for a 1 per cent. suspension of corpuscles. The serum of each animal was tested on the corpuscles of a number of other animals in order to determine its hæmolytic power. Up to the present time over 300 such tests have been made. The serum obtained from tumor dogs is almost without exception possessed of hemolytic power. This is least marked in the early and active stages of tumor growth, more so in the broken down and softened tumors. It persists even in the dogs which have recovered of their tumors. The corpuscles of the tumor dogs manifest a much greater resistance to this hemolytic activity of the serum than do the corpuscles of other dogs. The resistance is not absolute in the test tube, but in dilutions which are just sufficient to demonstrate the hemolytic activity of the serum from tumor animals on normal corpuscles, the corpuscles from tumor animals remain intact. The serum of animals without tumors has almost without exception failed to show any hemolytic power and the corpuscles have not been resistant to the serum derived from tumor dogs.

The characteristics of this hemolytic substance in the serum of tumor dogs have been the subject of further study. The serum loses some, or only little of its power, by being passed through a Berkefeld filter. Heating it to 85° for one hour does not destroy its activity. It differs markedly from the immune bodies known as amboceptors. It resembles in certain respects the hemolytic substances derived by extraction from necrotic tumors.

23 (279)

On the circulation through the kidneys. I. On vaso-motor reactions. II. The renal blood flow in relation to the pressure in the ureter and bladder. III. The effect of solutions of adrenalin.

By **R. BURTON-OPITZ** and **D. R. LUCAS.**

[*From the Physiological Laboratory of Columbia University, at the College of Physicians and Surgeons.*]

The experiments embodied in this abstract deal quantitatively with the renal blood flow, under different experimental conditions. They were performed upon dogs with the stromuhr described by Burton-Opitz.[1] The right and the left renal veins were used.

Besides the quantitative data, the authors succeeded in obtaining vaso-motor effects on stimulation of the præ, as well as post ganglionic fibers, the constrictory effects being in both cases the most prominent. The constriction of the blood vessels of the kidney was betrayed by a decrease in the venous return from this organ and a fall in venous blood pressure, this change being preceded by a brief increase of flow.

Among the post ganglionic fibers (renal plexus) a nerve was isolated which gave decided vaso-constrictory results.

In another series of experiments the pressure in the ureter was increased while the blood-flow in the corresponding renal vein was being recorded. The pressure was increased by means of air led into the ureter in the immediate vicinity of the kidney and of the bladder. Every increase in pressure from 20–120 mm. Hg resulted in a decrease in the venous return from the kidney and a

[1] Burton-Opitz: Archiv. f. d. ges. Physiologie, 1908.

fall in venous pressure, the decrease being in harmony with the height of the ureter pressure. A pressure of 20 mm. Hg or less remained ineffective when introduced next to the bladder, but produced a weak retardation of the venous flow when led into the upper portion of the ureter.

On increasing the pressure within the bladder, no effect upon the renal circulation could be noticed, not even when the organ was inflated until it burst.

By injecting solutions of adrenalin into the renal vein centrally to the stromuhr, a retardation of the venous flow was produced. The retardation appeared after an interval of from 7–9 seconds. The experiments speak against the presence of vaso-motor nerves in the central veins.

24 (280)

Some data regarding the portal circulation.

By **R. BURTON-OPITZ.**

[*From the Physiological Laboratory of Columbia University, at the College of Physicians and Surgeons.*]

The stromuhr referred to in the preceding abstract was used in obtaining quantitative data regarding the portal circulation. The experiments so far performed have given an average flow of 1.56 c.c. per second in the splenic vein (weight of spleen 78 grams) and a flow of 1.05 c.c. per second in the mesenteric.

In the case of the spleen, the veins draining the pyloric end of the stomach and fatty tissue of this region were ligated. The stromuhr was then inserted centrally to the last branch draining this organ. The venous pressure was recorded centrally to the instrument. In the case of the mesenteric the stromuhr was inserted distally to the point of entrance of the vena gastro-lienalis.

The nerves innervating the spleen and intestines were stimulated electrically while the blood flow in the veins was being measured. For the stimulation were selected first of all the entire bulk of the præ or post ganglionic fibers, and later on the most prominent fibers of the post-ganglionic paths.

On stimulation of the post-ganglionic fibers innervating the

spleen (splenic plexus) vaso-constrictory effects of a very pronounced character were obtained. Immediately on stimulation the flow in the splenic vein showed an extraordinary increase which soon gave way to an equally pronounced decrease. The flow regained its normal value sometime after the cessation of the stimulation.

The primary increase in the flow is not due to a vaso-dilation of the splenic blood vessels, but to a squeezing out of the blood "resident" in the spleen. Not until this amount of blood has been expelled by the constricting blood-vessels, can the decrease become evident. Thus, it appears that constrictory effects are obtained from the first when the nerves aforesaid are stimulated.

Although I have tested the vaso-motor power of several of the fibers composing the plexus, I have succeeded in obtaining only vaso-constrictory results.

Similar constrictory effects were incited by stimulation of the præganglionic fibres contained in the left splanchnic nerve.

Vaso-constrictory effects as displayed by a decrease in the flow and a fall in the venous pressure, were also obtained in the mesenteric vein on stimulation of the fibers centrally and distally to the ganglion mesentericum. Upon the evidence contained in the curves, the vaso-motor variations in the flow of the portal vein are said to be of peripheral origin. The portal vein itself possesses no vaso-motor mechanism.

25 (281)

A clinical viscosimeter.

By **R. BURTON-OPITZ.**

[*From the Physiological Laboratory of Columbia University, at the College of Physicians and Surgeons.*]

A pipette is used having a length of 20 cm. and a diameter of about 0.7 mm. The capacity of the tube, measured from its tip to a point above its enlarged middle portion, amounts to 1 c.c.[1] The tube is connected with a suction-pump developing from 50–100 mm. H_2O ; a T tube is interposed so that the pressure can be measured by means of a water manometer.

[1] Smaller tubes can be used, if the amount of fluid is limited.

The fluid, the viscosity of which is to be determined, is drawn into the pipette from a receptacle. By means of the usual method the time is recorded which elapses between the dipping of the pipette into the liquid and the moment when the fluid passes the mark above the bulb. A comparison is then made between this value and the value previously obtained for distilled water, the latter being regarded as 1. The experiments are performed at room temperature.

26 (282)

Studies in experimental arteriosclerosis.

By **Isaac Adler** and **O. Hensel.**

[*From the Laboratory of Dr. Isaac Adler, New York City.*]

Some time ago we reported to this society [1] that the intravenous injection of nicotin produced in the aorta of rabbits lesions practically identical with those obtained by Josue and others with intravenous injections of adrenalin.

The following is a brief summary of our further experiments along those lines. Many observers have laid particular stress upon the fact that where macroscopic lesions are not found, minute microscopic changes, stretching of the elastica and the like, could be recognized. We have counted as positive only such where there were distinctly visible macroscopic lesions. All observers agree that the best results can be obtained with older animals weighing over 2,000 grams. It was our misfortune that the great majority of rabbits obtainable by us were young and under the weight mentioned.

Twenty-four rabbits were treated with intravenous injections of nicotin, the dose being 5 drops of a 1–200 solution of pure nicotin. 7 died before they had had a sufficient number of injections. Of the remaining 17, results were positive in 7 after 18 to 77 injections. In 10 there were no macroscopic lesions after 20 to 117 injections.

In 6 rabbits nicotin and euphthalmin were used. Five were negative but one which had received alternate nicotin and euphthalmin injections 69 times showed aneurysm and calcification.

[1] Adler and Hensel: This journal, 1905, iii, p. 36.

The euphthalmin did not alter the typical nicotin reaction, such as convulsions, etc.

Nine rabbits received intravenous injections of nicotin and subcutaneous injections of iodipin. All were negative.

Two rabbits received their nicotin subcutaneously, the dose being double that given intravenously. One died too soon; the other was killed after 25 injections. Neither showed any arterial lesions.

One rabbit was given nicotin simultaneously with 1/10 grain atropin. It died after 14 injections; negative.

One nicotin and amyl nitrite; negative. Convulsions in no way influenced by these drugs.

Four rabbits were given nicotin and sodium nitrite; 3 negative and one positive after 49 injections. Two in which nicotin was injected into the peritoneum died after a few injections from some infection.

Two were fed by mouth with nicotin solution commencing with 5 drops and gradually increasing to 60. They were killed after 68 feedings. Result was negative.

Our series comprises 52 rabbits in all with 9 positive results. Excluding the 9 rabbits treated with iodipin and not counting all those experiments in which the animals died or were killed before a sufficient number of injections could be given, 23 in all, there remain 29 negative to 9 positive results.

Eight rabbits were treated with adrenalin of which 5 died after 1 to 8 injections; the other 3 after 29, 63 and 82 injections showed aneurysms and calcifications.

Five rabbits were treated with barium chlorid, 5 to 10 minims of a 1 per cent. solution. Two of these died after 3 injections, possibly from over-dose; the other three after 40, 65 and 69 injections were negative.

Two received barium chlorid and iodipin. One died after 2 injections from over-dose. The other, after 61 injections, showed no lesion.

One rabbit received 63 injections of euphthalmin with negative result. The dose, 3 milligrams, was probably too small.

Two treated with intravenous injections of calcium chlorid after 74 injections showed no lesion.

Two which were given digalen intravenously were negative after 52 and 64 injections.

Four were given acetate of lead intravenously. In two of these, by using a very dilute solution, 1–2,000, dose 30 minims, we managed to give 26 injections. Negative. Two others received subcutaneous injections which produced abscesses.

In the whole series of over 80 rabbits there are but 12 positive results.

Fourteen rabbits weighing considerably over 2,000 grams each, not subjected to any experimentation, were examined for aortic lesions with negative results.

A close scrutiny of the reports of other observers shows that they also have had many negative results. Adrenalin seems to give the best and most uniform results, though Kayserling in eight rabbits treated with adrenalin saw no macroscopic lesions and only a few microscopic changes. On the other hand, positive results have been obtained from substances differing most widely in chemical composition and physiological action, even from normal saline solution given in large quantities. On the whole it may be said that blood-pressure-raising drugs give the best results.

The extent and gravity of the lesions do not seem to be altogether dependent upon the number of injections given — Erb saw complete calcification of the aorta after a single injection of adrenalin, while we and many other observers have found very little or nothing after a great many injections. All attempts thus far to produce similar lesions in other animals, such as dogs and monkeys, have failed. The lesions seem to occur in rabbits, though probably but rarely, spontaneously and especially in such animals debilitated by old age and disease.

Much further investigation is necessary, but the following conclusions seem to us warranted in the present state of our knowledge :

It is still very uncertain what relation the experimental results produced in the aorta of rabbits by the intravenous injection of adrenalin, nicotin and other substances, may bear to human atheroma or arteriosclerosis. In the present state of our knowledge we are not justified in identifying the two processes. It seems probable that rabbits have a special predisposition towards these lesions. Young and healthy animals seem able to resist and render

innocuous the introduction of the various toxic materials employed. It is very probable that in animals debilitated by old age, malnutrition and disease, the aorta is no longer normal and is probably the seat of minute microscopic lesions such as stretching of the elastica and insufficiency of the muscular fibers. In such animals various toxins introduced into the circulation, especially those tending to raise blood-pressure, will bring out this latent disposition and cause arterial necrosis, calcification and aneurysmatic dilatation.

27 (283)

On the influence of various substances, applied directly to the medulla oblongata, upon the respiratory rhythm in frogs.

By **T. BRAILSFORD ROBERTSON.**

[*From the Rudolph Spreckles Physiological Laboratory of the University of California.*]

Recent experiments by Maxwell[1] have shown that the nerve cells in the cerebral cortex are not stimulated by the ordinary nerve stimulants, such as oxalates, citrates, tartrates, etc. On the other hand we are in the possession of the well-known fact that an increase in the CO_2 tension of the blood supplied to the brain at first accelerates and later, if the increase in CO_2 tension be sufficiently great, inhibits the rhythmic discharge of impulses from the respiratory center or centers. It appeared possible that although the ordinary nerve stimulants do not excite nerve cells yet some other group of substances might be found which does so. Accordingly the following experiments were undertaken.

The roof of the skull, in frogs, was removed by means of a fine pair of bone forceps as far down as the tip of the fourth ventricle; in some instances the membrane covering the floor of the fourth ventricle was removed, in others not. The cerebrum was removed by cutting across the thalamencephalon just in front of the optic lobes; by this means it was found that a more regular respiratory rhythm was obtained, the frog was quieter, and the results were more uniform. The cavity left in the skull by the removal of the

[1] Maxwell: *Journ. of Biol. Chem.*, 1906, ii, p. 183.

cerebrum was then plugged with absorbent cotton and absorbent cotton was placed all round the wound so as to absorb any fluid which might escape from the depression left by the removal of the roof of the skull. With practice the operation could be performed in 3 to 4 minutes, and, if successful, within 3 to 5 minutes after the operation respiration, which at first is suspended, is resumed. After about 5 to 10 minutes, the rhythm of respiration usually becomes constant and, if the frog be left untouched, remains so for over two hours.

After an interval of 15 minutes, to allow all shock effects to subside, drops of various solutions were placed upon the exposed surface of the medulla oblongata and their effect upon the respiratory rhythm noted.

It was found that N/10 solutions of acids (hydrochloric, sulphuric, lactic, oxalic) caused a notable acceleration. Thus in a frog breathing at the rate of about 19 respirations a minute the application of N/10 H_2SO_4 quickened the rate, within two minutes, to 50 respirations per minute. In other instances less acceleration was obtained, but in all cases it was very marked, except when the respiratory rhythm before the application of the acid was very convulsive and irregular in character owing to injury of the medulla during the operation; in these instances it is possible that only the spinal centers were functioning.

One per cent. KCN caused complete stoppage of respiration within three to five minutes. Strong reducing agents (1 per cent. formaldehyde, M/10 K_2S, M/10 hydroquinol, 3/4N sulphurous acid) caused marked slowing or stoppage of respiration. Oxidizing agents (20 per cent. Kahlbaum's C. P. H_2O_2, N/10 $KMnO_4$, N/10 Fe_2Cl_6) accelerated the rhythm but not markedly. Respiration was inhibited by N/10 $CuCl_2$ and slowed, but not markedly, by N/10 $HgCl_2$. Solutions of much higher osmotic pressure than the blood (*e. g.*, pure glycerol) moderately accelerate the rhythm and render it irregular; prolonged action of solutions of much lower osmotic pressure than the blood (distilled water, tap water) greatly slows the respiratory rhythm and may ultimately suppress it altogether. In confirmation of Maxwell's results I find that sodium oxalate has no effect upon the rhythm.

It may be questioned whether the effects observed are really

due to the direct action of the substances upon the respiratory center or whether they are reflex effects due to the action of the substances upon nerve fibers. If, however, we cover the lower limbs of the frog, treated as above, with a sheet of filter paper saturated with N/10 HCl, after 3 or 4 minutes of violent struggling the frog becomes quiet and the respirations, at first inhibited, are resumed; although occasional convulsive movements occur, these become less and less frequent and respirations continue except during the actual convulsions. The rate of the respirations is, however, unaltered for the first six minutes; in ten to twelve minutes they may be accelerated 10 per cent. but not more. Now the time which elapses before the respirations are quickened is only 1 to 2 minutes when the acid is applied directly to the medulla and the animal usually does not struggle at all. Moreover, the two effects, the effect upon the respiratory center and that upon nerve fibers can readily be distinguished from one another in the case of N/10 oxalic acid. At first the normal effect of an acid upon respiration is observed, namely a marked quickening of the rhythm so that after 10 minutes the rate may be doubled. If further applications of oxalic acid be made the rate continues to increase until over one half hour after the first application of oxalic acid when the muscles of the whole animal go into prolonged tetanic contractions; the former effect is that of the acid upon the respiratory center, the latter effect is that of the oxalic acid anion upon nerve fibers.

The results are such as to indicate that the processes occurring in nerve cells during the passage of a reflex are of the nature of oxidations and that they are accelerated by acids.

28 (284)

Metaplasia and metastasis of a rat tumor.

By **SIMON FLEXNER** and **J. W. JOBLING.**

[*From the Rockefeller Institute for Medical Research.*]

We reported to this society on several previous occasions some facts regarding a tumor of the rat which we have propagated for more than two years. This tumor is now in its twelfth genera-

tion. The original tumor was of complex histological structure and has been regarded by us as consisting predominantly of a tissue of sarcomatous nature and to a less extent of irregular tubular formations resembling structures seen in certain endotheliomata. In the fifth generation of one series of transplantations, metastases were first noted in the regional lymphatic glands. In all previous generations the metastases were to the lungs and the kidneys ; and in the other series this change in properties has not been observed. In the succeeding generations between the fifth and the eleventh, of the particular series mentioned, the lymphatic metastases have grown more common and more widely disseminated. Coincident with this change in properties of the tumor, a modification in the histological structure has been noted ; the tumor has become progressively more and more adenomatous in appearance until now it has entirely cleared itself of that part which had been taken to resemble sarcoma. Hence metaplasia of the tumor in the direction of carcinoma has taken place, with which change is associated the acquisition of the property of involving the nearby and distant lymphatic glands in its growth.

Twenty seventh meeting.

Schermerhorn Hall, Columbia University. February 19, 1908.
Vice-President Morgan in the chair.

29 (285)

An exhibition of photographs of chromosomes, with explanatory comment.

By **EDMUND B. WILSON.**

[*From the Laboratory of Zoölogy, Columbia University.*]

The speaker exhibited a series of lantern-slides from direct photographs, showing the sexual differences of the chromosomes in a number of species of insects. The facts have now been determined in nearly sixty species, all of which conform to the same principle that the spermatozoa are of two numerically equal classes, one of which is male-producing, the other female-producing. This is proved by the relations of the chromosomes. The two classes of spermatozoa show certain constant differences in this regard, differing in respect to one pair of chromosomes, and in a few cases in respect to two or three pairs. The somatic chromosome-groups of the two sexes show precisely parallel differences in every known case; and a study of the facts in detail proves that these differences must be due to the fertilization of the egg by one or the other class of spermatozoon. This conclusion, first reached by strictly cytological researches on the germ-cells of insects, has recently received complete experimental confirmation in the work of Correns on the diecious flowering plants.

30 (286)

The production of two kinds of spermatozoa in phylloxerans — functional "female producing" and rudimentary spermatozoa.

By **T. H. MORGAN.**

[*From the Laboratory of Experimental Zoölogy, Columbia University.*]

The work of McClung, Stevens, and Wilson has shown in the group of insects that sex-determination is associated with the presence of two kinds of spermatozoa — "male and female producing." From this point of view sex is determined by the sperm and not by the eggs in those species of insects in which parthenogenesis does not occur. Within the group of insects there are other species in which parthenogenesis appears as a part of the regular life-cycle. Such cycles are shown especially in the groups of aphids and phylloxerans. In these, all of the fertilized eggs produce females only, while from the parthenogenetic eggs both males and females develop. Hence it is evident that, in these groups at least, the egg may be sex-determining, but how this could take place has not been discovered.

In several species of Phylloxera that I have studied some facts have come to light that go far towards explaining sex-determination in this group. I shall describe first the spermatogenesis of a species that contains so small a number of chromosomes that the number can be counted accurately, not only in the reduced number of the spermatogenesis but in the somatic cells as well.

The reduced number of chromosomes is three. In the first spermatocyte division two of these divide equally, but the third lags behind the others, and finally in the very last stages of this division it retreats to one of the poles. Thus there are three chromosomes in one of the two daughter cells, and only two in the other. Still more significant is the fact that the cell with the fewer chromosomes is very small; it contains very little cytoplasm and subsequently degenerates without forming a spermatozoon. In the second spermatocyte division all three chromo-

somes of the larger cell divide equally, thus producing two spermatids with three chromosomes each. These spermatids become spermatozoa. They correspond in their mode of development to the "female-producing" spermatozoa of other insects. Hence we can understand why all of the fertilized eggs become females!

The question still remains as to how the males and the sexual females are produced from the parthenogenetic eggs. Here I have some observations to report which seem to indicate how this process takes place.

I find that the somatic cells of the males of the species referred to above contain only five chromosomes. These five give in the spermatogenesis the reduced number three by two uniting with each other and the third having no partner. I find that the somatic cells of the female contain six chromosomes. It follows that at some time in the life-cycle of the parthenogenetic eggs one chromosome disappears in those eggs that become males, while the full number is retained in the female. It seems plausible that this change takes place in the formation of the single polar body given off by the parthenogenetic egg.

The results seem to show that while the sex of the stem-mother is connected with the presence of "female-producing" spermatozoa, the production of males and of sexual females is dependent on a process that takes place in the egg analogous to the same process that takes place in the spermatogenesis of other kinds of insects. Hence it follows that the egg as well as the sperm has the power of determining sex by regulating the number of its chromosomes.

31 (287)

Physiological problems of the geographical distribution of Partula in Polynesia, with demonstration of specimens.

By **HENRY E. CRAMPTON.**

[*From the Department of Zoölogy, Columbia University.*]

The speaker described briefly the geographical features of the distribution of Partulæ in Polynesia, known through the researches of Mayer, Garrett, Cuming, and others, as well as from personal

observations. The snails of this genus are so distributed that each archipelago and each island where they occur possesses unique types, while often single valleys will comprise the habitat of a species. Only two exceptions to the former statement are known.

A detailed demonstration was made of the snails from 55 valleys of Tahiti, and from 19 valleys of Moorea, the two islands of the Windward division of the Society group. The present communication consisted chiefly of a description of the features presented by the demonstrated valley populations. The general conclusions of the survey are (1) that there is a general correlation between geographical proximity or isolation on the one hand and specific resemblance or divergence on the other hand; (2) that some species (*e. g.*, *P. hyalina*) are wide-spread and relatively invariable, while other forms exhibit variations and mutations that seem to be the antecedents of fixed independent varieties of the future; (3) and that variation does not seem to be referable to environmental influences.

32 (288)

Note on the isolation of carnaubic acid from beef kidneys.

By **EDWARD K. DUNHAM.**

[*From the New York University and Bellevue Hospital Medical College, and the Rockefeller Institute for Medical Research.*]

A lipoid obtained from beef kidneys and having solubilities similar to those of Liebreich's "protagon," yields, on cleavage with alcoholic hydrochloric or sulfuric acid, an ester of carnaubic acid, which separates on cooling. From this ester the free acid may be obtained by saponification with sodium ethylate and decomposing the resulting soap with a mineral acid. The free acid and its ethyl ester are freely soluble in ether and chloroform; also, in hot alcohol, benzene, acetone, ethyl acetate or acetic acid, but separate from these solvents on cooling. The acid melts at 72.4°, the ethyl ester at 50°, both uncorrected.

Analysis of the acid. It was purified by fractional precipitation with magnesium acetate. The magnesium soap was decomposed with hydrochloric acid. The acid was recrystallized from acetone :

Weights in grams: substance, 0.1590: CO_2, 0.4556; H_2O, 0.1870

Percentage Elementary Composition

Found	Calculated for $C_{24}H_{48}O_2$
C 79.147	C 78.159
H 13.161	H 13.156
O 8.691	O 8.684

Analysis of the ethyl ester:

Weights in grams: substance, 0.1414; CO_2, 0.4079; H_2O, 0.1677

Percentage Elementary Composition

Found	Calculated for $C_2H_5\,C_{24}H_{47}O_2$
C 78.670	C 78.685
H 13.271	H 13.245
O 8.059	O 8.070

Analysis of the silver salt:

Weights in grams: substance, 0.5294; Ag, 0.1208

Percentage Content of Silver

Found	Calculated for $Ag\,C_{24}H_{47}O_2$
22.818%	22.703%

The carnauba acid obtained from carnaubic wax has the same percentage composition and is recorded as melting at 72.5°.

33 (289)

The change of corpuscle resistance in the blood of immunized animals, coincident with the formation of anti-bodies.

By **FREDERICK P. GAY.**

[*From the Pathological Laboratory of Harvard University.*]

In logical sequence to the studies on isohemagglutination of human blood, the results of which have been presented to this Society[1] and recently published in detail, have followed estimations of certain physico-chemical properties of the blood of normal and of immunized animals. In human bloods three rather definite groups were found as regards interagglutination of corpuscles, groups which correspond to relative differences in tonicity of the bloods in question. The method principally relied on in the estimation of differences in the molecular concentration of various human bloods was that of the relative susceptibility of the respective blood corpuscles to hemolysis by salt solutions of different concentrations, a method which has been employed by Hamburger and others as the most delicate for this purpose.

[1] *This Journal*, 1907, v. p. 14.

In normal rabbits and guinea-pigs the percentage of hemolysis in a salt solution of given concentration is found to be in surprising correspondence between individuals of the same species. The mean isotonic solution in twenty two determinations of rabbit blood, comprising seventeen individuals, was found to be between 0.65 per cent. and 0.7 per cent. sodium chloride. In nine guinea-pigs the isotonic mean was found to be 0.1 per cent. salt solution lower than that of rabbits' blood (0.55 per cent. to 0.6 per cent.). The comparison of the freezing point of the whole defibrinated blood and of the blood serum of these two species, in several individuals, showed a corresponding difference in the freezing point of from 0.04°–0.06° C.

Rabbits were immunized with *B. typhosus* or *B. ozenæ* or the blood of guinea-pigs respectively; guinea-pigs were immunized with rabbit blood. In from nine to fourteen days after the last of several injections these animals were bled, the resistance of their corpuscles tested, and in many instances the freezing points of their defibrinated blood or of their sera determined. As regards the resistance of their corpuscles to salt solutions it was found that in every case it was increased in the immunized animal in an amount usually corresponding to a 0.1 per cent. NaCl solution. This relative difference in the resistance to hemolysis between the blood of normal and of immunized animals was controlled in as many ways as suggested themselves; animals were tested before and after immunization; normal animals were tested several different times; and in each instance normal and immunized animals were tested at the same time. In every instance the relative difference between normal and immunized animals would seem as definite as is indicated by the mean results.

In order to assert that this increased resistance of corpuscles in the blood of immunized animals to hypotonic solutions indicates a lower tonicity of the blood as a whole, it would be necessary to determine a corresponding difference in the molecular concentration of the blood or sera by freezing-point determinations. A considerable number of such determinations have been made but with results not wholly in accord with the corpuscle resistance method. In perhaps the majority of instances an actually higher freezing point (lower tonicity) is obtained for the immunized animals.

A large number of comparative tests are called for, before a definite statement can be made as to whether the increased resistance of the blood corpuscles of immunized animals is due to a change in tonicity of the blood as a whole or simply to a variation in the resistance of the corpuscles.

34 (290)

Further observations on the precipitation of inorganic colloids by sera.

By **CYRUS W. FIELD.**

[*From the Laboratory of the Department of Health, of New York City.*]

At the meeting of this Society last June, I recorded some facts which seemed to show that in rabbits the precipitating effect of their sera was greater for collodial platinum and collodial silver, after they had received injections of these two substances. Further work since that time on rabbits and on various sera from horses has shown that there are wide variations in the agglutinating or precipitating value for these colloids; not only in various animals but the value differs in the same animal at different times.

In a few cases in which I have been able to test the electrical conductivity of the sera, I have found that some of those which gave the highest conductivity gave the highest agglutinating effects, and, therefore, I believe that the variations in the agglutinating or precipitating effect of the sera is due to variations in the concentration of electrolytes. These inorganic colloids are, as is well known, extremely susceptible to the influence ef electrolytes and a very slight increase in the concentration of univalent kations and even more especially of the di- and trivalent kations, would cause wide variations in precipitating value. For instance, there might be a greater concentration of one divalent kation and a lessened one of some univalent kation and yet the total concentration of all electrolytes remain nearly the same.

35 (291)

A note on anaphylaxis.

By **EDWIN J. BANZHAF** and **L. W. FAMULENER.**

[*From the Research Laboratory of the Department of Health, New York City.*]

Rosenau and Anderson in their first communication on hypersusceptibility reported their attempts to destroy or remove the toxic substances from horse serum. They treated the serum, which was used for the second injection, by various physical and chemical means, such as, heating the serum to 60° C., filtering through porcelain, drying, freezing, precipitation and dialysis, and by direct addition to the serum for various lengths of time of such chemicals as potassium permanganate, hydrogen peroxide, succinic acid and butyric acid, also various antiseptics, all without success. Continuing their investigations they reported, in their second communication, the influence of ferments, alkaloids, salts and such substances as ox bile, animal charcoal and yeast cells. These gave negative results.

One of us took up the problem from another standpoint, *i. e.*, to treat the sensitized animal with drugs just prior to the second injection. In the preliminary experiments morphin sulphate was used with negative results. Following this, chloral hydrate was employed to produce hypnosis and it was found that sensitized guinea-pigs could be protected with this drug. After our experiments with chloral hydrate were well advanced, Besredka's communication came to our attention, in which he claimed that ether and also calcium chloride exerted a protective action when administered to sensitized guinea-pigs before the second injection of the serum.

We found by injecting a solution of chloral hydrate which was just sufficient to produce hypnosis, that fully 75 per cent. of all serum sensitized guinea-pigs were completely protected from the second injection of serum into the peritoneal cavity, while 90 per cent. of all the controls died. We believe that with improved technique on the dosage of chloral hydrate, we will be able to

protect 90 per cent. of all fully sensitized guinea-pigs. By fully sensitized, we mean, that three weeks or a month should elapse before the second injection of serum into guinea-pigs which have survived the routine testing of antitoxin. For guinea-pigs which have received horse serum alone ($\frac{1}{100}$ to $\frac{1}{500}$ c.c.), at least 7 or 8 weeks should elapse before the second injection. By allowing the above elapse of time, over 90 per cent. of our controls died within an hour, most of them within 20 minutes.

We have found that the dose of the chloral hydrate per gram weight of the animal was more or less variable; no fixed amount can be stated, much depending upon the individual idiosyncrasy of the animal. Approximately 75 milligrams of the drug to a 250 gram guinea-pig and 100 milligrams to a 300 gram guinea-pig produce the degree of hypnosis desired.

We use a fresh 10 per cent. solution of chloral hydrate. We carefully measure out the required amount into a small sterile beaker and add an equal amount of sterile water. This diluted solution is injected into the muscles of the thigh of the animal, half into one leg and half into the other. After an elapse of 20 to 30 minutes the needle is inserted into the peritoneal cavity; muscular twitching and slight movement of the head will be noticed. The injection of 5 c.c. is then given and the animal kept in a warm room. No symptoms appear and the sleep is undisturbed. After an elapse of 1–½ to 2½ hours the animal will slowly recover from the effects of the drug. No symptoms or ill effects have been observed in any of the animals. Observations have been followed for over two weeks after treatment. The animal, after the effects of the drug have disappeared, is immune to a third injection of serum, until resensitized, which will be after an elapse of 2 to 2–½ months for slight sensitization and 3 to 4 months for full sensitization.

If the dose of chloral hydrate has not been sufficient the insertion of the needle into the peritoneal cavity will cause pronounced muscular movements, the raising of the head and an attempt to regain its feet. Under these conditions if the serum is injected the animal will die of anaphylaxis.

On the other hand, if the animal shows no muscular twitchings the dose of chloral has probably been too large.

We wish to emphasize the fact that great care must be used not to overdose the sensitized guinea-pig with chloral hydrate. A sensitized, as well as a normal guinea-pig, will recover from a large dose — considerably more than the amount mentioned above. But, apparently, the combined effects of the drug and the serum in a sensitized animal produce a deeper hypnosis than the drug when given alone.

Thus far, our experiments have been only with the intraperitoneal injection. Besredka's method of injecting directly into the brain will be taken up later and also the method of injection directly into the vessels according to Gay and Suthard and into the heart according to Lewis.

In a number of experiments with calcium chloride which Besredka claims to have a protective action, we were unable to save the sensitized animals. They died with characteristic symptoms of anaphylaxis.

We have also failed to substantiate Besredka's claims that sensitized guinea-pigs under the influence of ether narcosis are protected from the second injection of ¼c.c. of serum into the brain. Neither does ether narcosis protect them when the injection is made directly into the vessel or directly into the heart. All the animals died with characteristic symptoms of anaphylaxis in from 2 to 6 minutes. Normal control guinea-pigs under the influence of ether narcosis showed no symptoms or ill effects when subjected to injections of physiological saline solution by the above methods.

In conclusion, we wish to add that, other chemical substances belonging to the same group as chloral may show similar action in protecting guinea-pigs against anaphylaxis.

36 (292)

The relation of plasticity to sex and age in the dancing mouse.

By **ROBERT M. YERKES.**

[*From the Psychological Laboratory of Harvard University.*]

Plasticity or the modifiability of behavior in the dancer has been studied by measurement of the rate of habit formation. The

habit of choosing a white box instead of a black box was established by means of systematic training, on the basis of the association of an electric shock with the latter. Each animal was subjected to ten tests daily until it chose correctly on three consecutive days. The habit was then considered perfect and the training ceased. We may speak of the number of tests necessary for the establishment of a perfect habit as the index of plasticity. The index, then, may be defined as the number of tests up to the point at which errors of choice ceased for at least three days.

In order to discover the relation of sex and age to plasticity five pairs of mice were trained for each of the ages, one, four, and seven months. The investigation is unfinished, as older individuals are to be tested.

TABLE: INDICES OF PLASTICITY.

Age.	Males.	Females.	Both.
1 month	82	106	94
4 months	128	106	117
7 months	192	146	169

In this table each index for the males and the females is the average for five individuals. In the last column the results for the sexes are combined.

Sex differences which appear in the above table.

1. At the age of one month the males learn more quickly than do the females. The males require only 82 tests; the females require 106 for the establishing of a perfect habit. Consequently the index of plasticity for the one-month males is 82; that for the one-month females, 106.

2. At the age of four months the opposite is true. The females learn much more quickly than the males. As the table shows, the index for the males is 128; that for the females 106.

3. At the age of seven months a similar relation holds.

Age differences which appear in the above table.

1. From one month to seven, at least, the plasticity of the male dancer steadily and rapidly decreases.

2. There is little change in the plasticity of the female dancer between the first and the fourth month. If anything the rapidity of learning tends to increase. Between the fourth and seventh

month a sharp decrease in plasticity occurs as is indicated by the rise of the index from 106 to 146.

3. The racial indices of modifiability, obtained by averaging the results for five individuals of each sex for each of the three ages mentioned, indicate a rapid decrease in plasticity from 94 at one month to 169 at seven months of age.

Discussion of these results is not in order in the present unfinished state of the investigation. I may say, however, that I am attempting to ascertain the duration of the sexual life of the dancer, and to discover the relation of plasticity to the strength of the electric stimulus which the animal learns to avoid.

37 (293)

The crystallography of hemoglobins.

By **EDWARD T. REICHERT** and **AMOS P. BROWN**.

[*From the S. Weir Mitchell Laboratory of Physiology, University of Pennsylvania.*]

The primary object of this research was to determine whether or not corresponding albuminous substances are identical. We believed that if non-identity were established we should have a fact of great fundamental importance in relation to heredity, the origin of species, etc. The crystallographic method was adopted because we believed that by this means we might succeed where the chemist has failed; and hemoglobin was selected as the first substance of our inquiry because of its comparatively ready crystallizability. Crystals from over one hundred species have been studied, and we have not only positively shown the non-identity of the hemoglobins, but have also brought to light much information of broad biological interest. From a preliminary review of our records certain facts stand out very prominently.

1. *The constancy of generic characters in the crystals.* — For instance, the genera that have been represented by a considerable number of species, as *Felis*, *Canis*, *Papio*, etc., show in each case constancy in the characters of the crystals in each genus, with marked differences both in axial ratios and in crystal systems between the

crystals from the different genera. When the characters of the crystals from any one of these genera are tabulated, they at once recall to the mineralogist the crystallographic groups of minerals. The crystals of the genus *Felis* are as strictly isomorphous as those of the calcite group of the rhombohedral carbonates. As an example of the individuality of these generic characters, the following example may be cited: A sample of blood, marked as that of a certain species of baboon was received from one of our zoölogical gardens. Upon making preparations and examining the crystals, it was at once evident that they did not correspond to any species of baboon thus far examined, nor did they show the characters of the crystals of this genus. They were identified by their crystallographic characters as belonging to the cats (genus *Felis*), but not to any species thus far examined. Inquiry at the zoölogical garden from which the blood was received showed that the animal recorded as being subjected to a postmortem examination on the date when the blood was collected was a species of cat (genus *Felis*), but not one of which we had previously examined the blood.

2. *Specificity in the crystals of a genus.* — The crystals of different species of a genus can usually be distinguished from each other by definite differences of angle, when they are favorable for good measurement, while preserving their isomorphous character as belonging to a definite genus. From the difficulty of measurement in many cases it is hard to give these differences a quantitative value, but the variation in habit of the crystals and their mode of growth will often show specific differences.

3. *The occurrence of several types of crystals of oxyhemoglobin in many species.* — The oxyhemoglobin of some of them is dimorphous, crystallizing in two systems, or even trimorphous in many cases. Here it is generally seen that the crystals first formed crystallize in a system of a lower grade of symmetry than those formed later. When several types of crystals occur in the species of a genus, they may each be arranged in isomorphous series. The explanation of these observations seems to be indicated by the results obtained.

4. *The constant recurrence of certain angles, either plane or dihedral, in oxyhemoglobin, hemoglobin, and the "methemoglobin" of various species, even when widely separated zoölogically and when*

the crystals belong to various systems. This indicates a common substance in hemoglobin or a common structure in the various hemoglobin molecules.

5. *The importance of twinning in the formation of crystal aggregates and the constant recurrence of certain types of twinning in all of the hemoglobins.* These results will likely throw light upon the mechanism of twinning and be of importance in general crystallography.

6. *Differences between oxyhemoglobin and hemoglobin or reduced hemoglobin, in certain species.* Undoubted differences between the crystals of these two substances in the crystals of the same species have been observed.

We have gathered additional evidence leading to the conviction that other corresponding proteins, as well as certain fats and carbohydrates, will be found to exhibit similar generic specificities.

Our first memoir on this subject will shortly be published by the Carnegie Institution of Washington, under whose auspices this research is being conducted.

38 (294)

The germicidal property of milk.

By **M. J. ROSENAU** and **GEO. W. MCCOY.**

[*From the Hygienic Laboratory, U. S. Public Health and Marine Hospital Service, Washington, D. C.*]

Judged by the number of colonies that develop upon agar plates the bacteria in milk first diminish, then increase in number. The so-called germicidal property of milk occurs only in the fresh raw fluid.

For the most part our work plainly shows that no actual reduction in the number of bacteria occurs. However, when compared with the controls a restraining action is evident. The phenomenon, therefore, appears to be that of a weak antiseptic rather than that of a true germicide.

When milk is kept warm (37° C.), the decrease is pronounced within the first eight or ten hours. After this time the milk has entirely lost its restraining action.

When the milk is kept cool (15° C.) the decrease is less marked, but more prolonged.

The decrease in the number of bacteria is largely apparent, being due at least in part to agglutination.

The bacterial clusters may, to a certain extent, be shaken asunder. This fact goes far to reconcile the discordant results of the various investigations upon the germicidal properties of milk. Those who used dilution methods with vigorous agitation broke up the bacterial clusters and thus obtained a larger number of colonies upon agar plates than those who plated directly with different technique.

Some of the polymorphonuclear leucocytes in milk seem to possess the power of phagocytosis, judged by microscopical preparations. Phagocytosis, however, plays no essential part in the "germicidal" action of milk, for the decrease in numbers is quite as marked in the cell-free serum as in the sediment rich in leucocytes.

The germicidal action of milk is specific. For instance, one sample restrained typhoid and *Staphylococcus pyogenes aureus* but not paratyphoid *A* or *B*.

Dilution experiments demonstrate the enfeeblement of agglutinins rather than the presence of a germicidal substance in solution.

The germicidal actions of blood and milk resemble each other in many particulars. The difference is largely one of degree. Blood serum acts more quickly and more powerfully than milk.

Freezing milk for ten minutes does not affect the phenomenon in question. In one experiment, freezing for 48 hours did not influence its restraining action upon typhoid, but destroyed it for *B. lactis aerogenes.*

Boiling milk or heating it above 80° C., destroys its "germicidal" properties. The effect of lesser degrees of heat varies with the microörganism. Thus, the restraining action for *B. lactis aerogenes* is weakened at 55° C., and almost destroyed at 60° C.; for typhoid it is not affected by heating the milk at 60° C. for twenty minutes, but is materially influenced at 70° C. for thirty minutes.

The "germicidal" action of milk varies in different animals and in the milk from the same animal at different times. At most, the action is variable and feeble. It cannot take the place of cleanliness and ice, but may be taken advantage of in good dairy methods.

Twenty eighth meeting.

Physiological Laboratory of the New York University and Bellevue Hospital Medical College. April 15, 1908. President Lee in the chair.

39 (295)

Influence of cold and exercise in phlorhizin glycosuria.

By GRAHAM LUSK.

[*From the Physiological Laboratory of the University and Bellevue Hospital Medical College.*]

When phlorhizinized fasting dogs with a urinary D : N ratio of 3.65 : 1 are exposed to cold they at first lose extra sugar which is derived from body glycogen. But if the exposure to cold be repeated the D : N = 3.65 : 1 may remain unaltered.[1] The lowering in environmental temperature was such as would increase fat combustion in the animal by 50 per cent. and yet the sugar output remained unchanged.

Mechanical work at first brings about an increased sugar excretion. If, however, a dog be freed from glycogen mechanical work has no influence on the sugar excretion. In the following experiment a fasting dog was made use of on the third day of total phlorhizin glycosuria. He was prepared by administering cold baths on the first and second days of the glycosuria, and then exposing him to a temperature of 10° C.; shivering removed his surplus glycogen. The mechanical work was done in a wheel during five-minute intervals of alternate work and rest throughout a first hour of a two-hour period. The results were as follows:

Period in Hours.	Distance Travelled in Meters	D	N	D : N
2		4.20	1.19	3.53
2	1,500	5.32	1.36	3.90
2		4.57	1.26	3.63
2	1,500	4.62	1.26	3.67

[1] Confirms Brasch, Minkowski, Allard; opposes Lüthje.

The fat metabolism during the hour of work must have been double that of an hour of rest, and yet in the last experiment there is absolutely no change whatever in the sugar excretion as a result of travelling 1,500 meters. The D : N ratio is therefore absolutely independent of fat metabolism, but is dependent upon protein metabolism.

To illustrate the manner of sugar production from protein, glutamic acid with its five C atoms was administered subcutaneously and *per os* to a phlorhizinized dog. The resulting increase in the output of urinary sugar was such as would indicate certainly a conversion of three and possibly a conversion of four of the carbon atoms of glutamic acid into dextrose. One can explain the former case as a result of the cleavage of glutamic acid into an alanin radicle which is convertible into lactic acid in metabolism and this again into dextrose.

The writer was assisted in this work by Mr. H. P. Mencken.

40 (296)

The influence of carbohydrate on the protein metabolism of a fasting pregnant dog.

By **J. R. MURLIN.**

[*From the Physiological Laboratory of the University and Bellevue Hospital Medical College.*]

A dog in the ninth week of pregnancy and weighing 12.46 kgm., on the third fasting day of a three-day period was fed 42 gm. of cane sugar for two days. The reduction in the nitrogen elimination on the second sugar day as compared with the last fasting day was over 50 per cent. The same experiment was repeated on the same dog more than two months later, *i. e.*, four weeks after the puppies were weaned. The dog weighed on the third fasting day 10.42 kgm. Since the puppies when they were born (four days after the conclusion of the former experiment) weighed 1.5 kgm., this probably represents, as nearly as one can estimate, the weight of the mother alone at the time of the former experiment. The cane sugar fed, therefore, would represent about the same percentage of the actual requirement on the part of the

mother dog in both cases. The reduction of the nitrogen elimination on the second sugar day as compared with the fasting day just preceding, was 20 per cent. in the non-pregnant condition instead of 50 per cent. in the pregnant condition.

TABLE I.

DOG PREGNANT 9TH WEEK.

Date, Dec., 1907.	Weight, Kgm.	Carbohydrate.	Nitrogen in the Urine.													
			Total.	Urea.		NH_3		Urea+NH_3		Creatinin.		Creatin.		Undetermined.	
				Gms.	Per Cent.	Gms.	Per Cent.	Gms.	Per Cent.	Gms.	Per Cent.	Gms.	Per Cent.	Gms.	Per Cent.
7	12.46	3d day fasting.	5.036	4.383	87	0.190	3.7	4.573	90.7	0.078	1.5	0.084	1.6	0.301	6.2
8	12.24	42 gm. cane sugar.	3.401	2.614	76.9	0.120	3.5	2.834	80.4	0.082	2.4	0.141	4.1	0.344	13.1
9	12.10	42 gm. cane sugar.	2.495 (50% red.)	1.874	75.1	0.058	2.3	1.932	77.4	0.085	3.4	0.164	6.5	0.314	12.7
10	12.00	fasting.	3.240	2.627	81.0	0.137	4.2	2.764	85.2	0.050	1.5	0.157	5.4	0.269	7.9

TABLE II.

SAME DOG, FOUR WEEKS AFTER WEANING PUPPIES.

Date, Feb., 1908.	Weight, Kgm.	Carbohydrate.	Nitrogen in the Urine.													
			Total.	Urea.		NH_3		Urea+NH_3		Creatinin.		Creatin.		Undetermined.	
				Gms.	Per Cent.	Gms.	Per Cent.	Gms.	Per Cent.	Gms.	Per Cent.	Gms.	Per Cent.	Gms.	Per Cent.
15	10.42	3d day fasting.	2.856	2.420	84.7	0.133	4.6	2.553	89.3	0.089	3.1	0.045	1.6	0.169	6.0
16	10.20	42 gm. cane sugar.	2.520	2.150	85.3	0.168	6.6	2.318	91.9	0.078	3.1	0.055	2.2	0.069	2.8
17	10.16	42 gm. cane sugar.	2.284 (20% red.)	1.942	85.0	0.074	3.2	2.016	88.2	0.080	3.4	0.059	2.5	0.129	5.9
18	10.04	fasting.	2.352	1.999	85.1	0.084	3.5	2.083	88.6	0.081	3.4	0.040	1.7	0.148	6.3
19	9.82	fasting.	2.587	2.127	82.2	0.124	4.8	2.251	87.0	0.081	3.1	0.056	2.2	0.199	7.7

The greater effect of the sugar in the former experiment is due to a large relative, as well as an absolute, reduction in the urea plus ammonia nitrogen. The explanation might be either that the sugar interfered with the production of urea and ammonia, that is, with deamidization and dehydration of the proteins placed in circulation when fasting is superimposed on the pregnancy, or that

the sugar has helped in the utilization of such proteins for the maintenance of the fœtal growth. The latter is, I think, the better interpretation. It would seem that the sugar has diverted substances which would otherwise have been eliminated as urea or ammonia, and it seems probable that this has been accomplished by synthesis in the embryonic tissues. The very high creatin nitrogen eliminated in the pregnant condition may be taken as an indication that the muscles are the chief source of these proteins but that the creatin itself is not (all) available for the embryonic growth. The fact that the dog was apparently weakened much more by this fasting period than by the period of similar duration when not pregnant would lend support to this view. The conditions would be entirely analogous, therefore, to Miescher's classical case of the fasting Rhine salmon where it has been shown that the muscular tissues are levied upon for the growth of the germ cells just previous to the spawning season. At all events it is clear from the above experiment that the carbohydrate has caused a much greater retention of the proteins in the pregnant condition.

Perfectly concordant results were obtained on a second dog in the sixth week of pregnancy where the reduction in the nitrogen output by a proportional amount of carbohydrate was 38 per cent.

41 (297)

The transplantation of parathyroid glands in dogs.

By **W. S. HALSTED.**

[*From the Hunterian Laboratory, Johns Hopkins University.*]

Our experiments, begun in the winter of 1906–7, have with interruptions been continued to date. In the course of the work many questions have arisen which still require solution and we find ourselves on the threshold of the investigation.

The first attempt of which I know to transplant these organs was made by us in December, 1906. Two parathyroid glands, one from the right and one from the left side of the dog's neck, were successfully implanted into the thyroid lobes from which they were removed.[1]

[1] Halsted: *American Journal of the Medical Sciences*, 1907, cxxxiv, No. 1 (July).

Leischner[1] succeeded in a small percentage of his cases in transplanting præperitoneally parathyroids in rats. These were autotransplantations, the donor being the donee. Pfeiffer, Hermann and Mayer[2] made two successful autotransplantations in puppies.

Beidl, commenting on the unsuccessful attempts of Foges, Kreidl and himself to transplant ovaries, testicles and suprarenal glands contrasts these failures with his success in the transplantation of the parathyroid glands. He states that a year prior to his reports,[3] he transplanted in two dogs, into the spleen, "foreign" parathyroids and "after a time" removed both thyroid lobes as well as the parathyroids. One animal lived seven months without a trace of tetany and finally died of what seemed to be "cachexia thyreopriva." The spleen contained, the report states, two well healed, intact parathyroid glands. The second dog had tetany of short duration. It recovered, however, entirely, still lives and consequently has, the author believes, parathyroids in the spleen which are functionally sufficient.

With the exception of the two cases of Beidl I find no report of the successful isotransplantation of the parathyroid glands, and, besides my own, the only successful autotransplantation of these glandules in dogs are, perhaps, the two reported by Pfeiffer, Herman and Mayer. As to the successful isotransplantations of Professor Beidl I confess to a little surprise in view of the facts that no deficiency was created before the transplantation and that both parathyroid glands survived in both cases. The absolute functional proof is lacking in these cases, inasmuch as the transplanted glands were not excised during life.

FIRST SERIES OF RESULTS (WINTER OF 1906–7). *Autotransplantation.* — Parathyroid deficiency is of necessity created in the autotransplantations. Of five autografts into the thyroid lobes of three dogs, three were successful (macroscopic and microscopic proof). Of eight autografts into the spleens of three dogs one only succeeded (macroscopic proof). In no instance was functional

[1] Leischner: *Archiv für klinische Chirurgie*, 1907, lxxxiv, p. 208.

[2] Pfeifter, Hermann and Mayer: *Mitteilungen aus den Grenzgebieten der Medizin und Chirurgie*, 1907, xviii, p. 377.

[3] Beidl: *Wiener klinische Wochenschrift*, Feb. 27, p. 304.

proof of the success of these transplantations obtained. Such proof cannot, of course, be so convincingly obtained in the cases of implantation to thyroid because of the lack of certainty that no parathyroid tissue except the transplanted remains at the time of the final operation, at which well nourished thyroid tissue, sufficient to insure the life of the transplanted parathyroid gland, must be left.

Isotransplantation. — In five cases (Dogs K, L, M, N and O), two, seven, five, five and eight parathyroids respectively were transplanted into the spleen. In only one dog (K) was a parathyroid deficiency created. In no instance was the transplantation successful; furthermore tetany supervened and death occurred just as promptly, after removal of the thyroid and parathyroids in the neck, in these dogs with so many intrasplenic isografts as in the ungrafted dog. Hence we may conclude that life is probably little, if at all, prolonged by the absorption of foreign parathyroids transplanted into the spleen.

SECOND SERIES OF RESULTS (WINTER OF 1907–8). — The transplantations were made, usually one gland at a time, at intervals of from seven to ten days, behind the rectus abdominis muscle, within its sheath.

Autotransplantation.—Of eighteen autotransplantations in twelve dogs, seven parathyroids were absorbed or necrotic (Dogs 3, 4, 5 and 10); five to seven lived and performed their function (Dogs 1, 7, 8, 9 and 11). What the fate of the four remaining glands would have been (Dogs 2, 3 and 14) is doubtful, the dogs having died of distemper. Four dogs are living and in good health, each, presumably, with only one autograft.

Isotransplantation. — Of twenty isotransplantations with created deficiency (Dogs 7, 12, 13, 15, 16, 17, 18, 19, 20 and 24) nineteen parathyroids were absorbed or necrotic. The result in one instance remains to be determined. Dog 7, deprived of all parathyroids except the one transplanted (an autograft), lived in good health and spirits twenty-five days or until at a third operation, the sustaining parathyroid was removed. There was in this dog, usually, a suggestion of hypoparathyroidism in a barely perceptible fibrillary tremor of the tongue and of the temporal muscles. On removal of the perfectly normal autograft behind the rectus muscle,

tetany developed within twenty-four hours and death occurred within forty-eight. Isotransplantation or isografting (two grafts) was unsuccessfully resorted to 24 hours after the supervention of tetany.

Summary. — Our experiments have proved that in dogs

1. Parathyroid glands are essential to the life of the animals, and that tetany follows their removal.

2. Transplanted parathyroids (autografts) may for an undetermined time perform at least the most evident function of these bodies.

3. One successfully transplanted parathyroid may suffice to maintain a fair degree of health; traces of hypoparathyroidism may persist.

4. In autotransplantation success is more common than failure.

5. Heterotransplantation rarely succeeds.

6. For the successful transplantation of these organs a deficiency of parathyroid tissue should be created.

7. Transplanted in excess of what is required by the organism parathyroid glands do not survive.

8. Excised or deprived of their blood supply in the course of an operation parathyroids should probably be reimplanted, preferably into the thyroid gland.

9. Complete excision of the thyroid lobes is well borne, for months at least, by these animals. Myxedema begins to manifest itself in a few weeks.

42 (298)

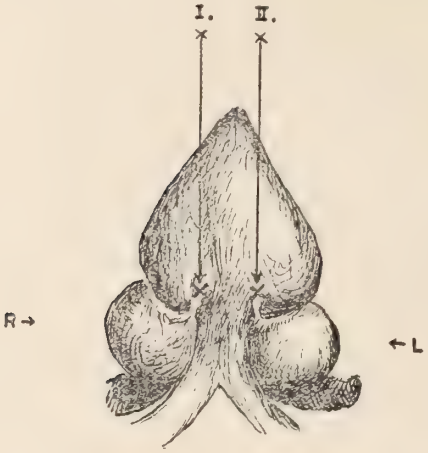
The nervous coördination of the auricles and ventricle of the heart of the lizard.

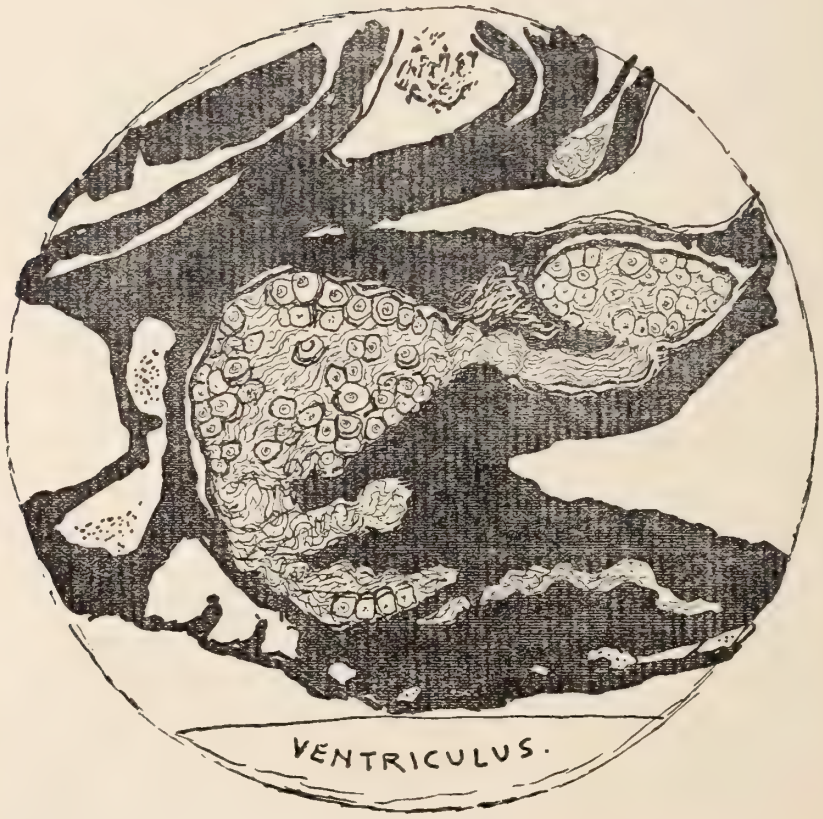
By **MARIE IMCHANITZKY.**

[*From the Hallerianum, Bern, Switzerland.*]

Communicated by S. J. Meltzer.

The following investigation was carried out on the hearts of lizards (*Ocellata lacerta*) for the purpose of studying the nature of conduction between the auricles and ventricles in these animals. The experimental part was done at the Zoölogical Station of


I.
II.
R→
←L


VENTRICULUS.

Villafranca. Ligatures were applied between auricles and ventricle and the pulsations counted before and after. The histological examination of stained serial sections was made later in the Physiological Institute of Bern.

An abbreviated protocol will illustrate the experimental results.

October 4, 1907. The beating heart of a large lizard whose brain was destroyed, was turned over within the open thorax so that the dorsal surface, where the chief nervous connections between auricles and ventricle are to be found, was lying upward. Whitish cords connect the auricles with the ventricle; the left cord is finer than the right and not always distinguishable. Besides these cords only connective tissue can be noticed between auricles and ventricle.

The heart was beating 25 times a minute, auricles and ventricle beating in regular sequence. After ligating the right cord the ventricle stopped beating for one minute, while the auricles continued to beat irregularly. Four minutes later the auricles were beating 18 times and the ventricle 10 times a minute. Later counts gave the following relations: 24 to 12, 24 to 18, 22 to 16. Twenty minutes after ligature the auricles were beating 25 times and the ventricle 22 times a minute. After ligating the left cord also, auricles and ventricle stood still for about one minute, when both began to beat. Again the relations were 10 to 3, 10 to 5, and 14 to 6.

Other experiments gave constantly similar results.

Kronecker and Spalitta observed in Palermo similar incoördination after applying ligatures to the heart of a sea turtle, but the results were not constant. (Report of the Berlin Academy of Science, May 25, 1905.)

In the hearts of lizards in which the cords were not visible, ligatures made through the corresponding part of the wall produced the same incoördination. Ligatures on the anterior (ventral) surface had no effect.

The histological examination showed that there are no traces of muscle bridges connecting the auricles with the ventricle. Neither is there in the septum a tract of muscles corresponding to the bundle of His. There are, however, a multitude of nervous connections between auricles and ventricle. The cord consists

of fine non-medullated nerve fibers intermingled with numerous large ganglion cells. There are also large capsules filled with nerve cells. Some of these oval-shaped capsules send out quite large nerve tracts, others give off only fine nerve fibers. With the intravital method of staining, fine nerve plexuses can be seen all over surrounding muscle fibers.

According to these studies the lizard heart may serve as a typical instance of a purely neurogenic origin of the coördination of the heart beats.

43 (299)

The influence of diet on the chemical composition of the body.[1]

By **LAFAYETTE B. MENDEL.**

[*From the Sheffield Laboratory of Physiological Chemistry, Yale University.*]

The investigation was an attempt to ascertain to what extent, if at all, the fundamental chemical structure of an organism can be altered by variations in diet or changed nutritive conditions. White mice were kept on dietaries of widely different character, *e. g.*, high protein, protein and fat, low protein and carbohydrate, etc., during considerable periods of time, and then killed and analyzed. The data are being published elsewhere. They are interpreted to indicate that although the fat and water content of such organisms show variations through a very wide range, there is a constant interdependence, even in cases of malnutrition. High content of fat is accompanied by lower water content, and *vice versa*. When the water content of the body is calculated on the basis of the fat-free tissue, the range of variation is remarkably small (70 to 74 per cent. of water). In order to afford some direct basis for a comparison of the tissue substance aside from its water and fat and the inorganic skeletal structure, the nitrogen content of the entire animals was calculated on a water-, fat- and ash-free basis. With few exceptions the animals afforded figures within narrow range above or below 16 per cent. of nitrogen. The constancy of composition of the organism suggests that it is

[1] This research was conducted with the aid of a grant from the Carnegie Institution of Washington.

not possible ordinarily to upset the relative composition of the body by dietetic measures, aside from altering the fat and glycogen content. Normal growth proceeds only through assimilation of all the essential body constituents in the proportion in which they are normally found in the body; and in tissue disintegration the loss is likewise general, not restricted to individual components of the fundamental structure.

44 (300)

The chemical composition of nonstriated mammalian muscle.

By **LAFAYETTE B. MENDEL** and **TADASU SAIKI.**

[*From the Sheffield Laboratory of Physiological Chemistry, Yale University.*]

In view of the paucity of data on the chemical composition of nonstriated muscle, Dr. Saiki has made an extensive study of the urinary bladder and muscular coat of the stomach of the pig. The details will be published in the *Journal of Biological Chemistry.* The preparations studied contained a considerably larger proportion of connective tissue (and presumably lymph spaces) than the corresponding skeletal muscles. This factor, involving the possible contamination with tissue lymph, must be taken into consideration in an interpretation of the analytical data. Hypoxanthin is the predominant purin base present. Creatin and paralactic acid can also be isolated. There is little, if any, glycogen in the nonstriated muscles examined; but the tissues possess the property of transforming glycogen in the characteristic enzymatic way. The most interesting contribution is a rather complete analysis of the inorganic constituents indicating a difference in their relative distribution in comparison with skeletal muscle, which can be accounted for in part only by an admixture of lymph.

COMPARATIVE COMPOSITION OF PIG'S MUSCLE AND BLOOD SERUM.

	K_2O	Na_2O	Fe_2O_3	CaO	MgO	Cl	P_2O_5	H_2O
Nonstriated muscle.....	0.081	0.328	0.011	0.044	0.007	0.171	0.184	80.6
Skeletal muscle (Katz)..	0.306	0.210	0.008	0.011	0.047	0.048	0.487	72.9
Blood serum (Abderhalden).....	0.027	0.425	—	0.012	0.004	0.363	0.020	91.8

The summary here given indicates a comparative richness of the nonstriated muscle in Ca. It is suggested that this may bear some relation to the characteristic physiological properties of such tissue, viz., tonic contraction and automatic rhythmic activity, both of which can be facilitated by Ca ions.

45 (301)

Increased susceptibility of protozoa to poison due to treatment with alcohol.

By **LORANDE LOSS WOODRUFF.**

[*From the Sheffield Biological Laboratory of Yale University.*]

The protozoa on which the experiments were performed were from two cultures (each comprising four lines) — one of *Paramecium* which had been under daily observations for over nine months, and the other of *Stylonychia* which had been under daily observation for over two months when the experiments were begun. The daily rate of division of each of these cultures was known.

From each of these cultures two secondary cultures were isolated line by line, and these were treated in identically the same way as the original or "control" cultures, except that each received daily for over a month a certain amount of alcohol in the culture medium of hay infusion. One culture received one part of alcohol to 2,500 parts of culture medium and the other received two parts of alcohol to 2,500 parts of culture medium.

Then, from each of the two control cultures and from each of the four alcohol treated cultures, other cultures were isolated and treated in identically the same way as the culture from which each was respectively derived, except that each received one part of copper sulphate to 1,250,000 parts of culture medium.

From these experiments it was found that whereas the average rate of division of the alcohol treated cultures was more rapid than that of the control, the alcohol treated cultures were more susceptible to copper sulphate than the control series, and finally (in the cultures carried to conclusion) died out while the control series treated with copper sulphate survived. It was found that the protozoa which were subjected to the greater strength of alcohol (2/2,500) divided more rapidly than those which were subjected to

the less strength and also were more susceptible to the influence of copper sulphate.

46 (302)

The relative specificity of anaphylaxis.

By **F. P. GAY** and **E. E. SOUTHARD**.

[*From the Pathological Laboratory of the Harvard Medical School.*]

The anaphylaxis in guinea-pigs caused by the previous injection of any one of the protein substances, horse serum, egg white, or milk is only relatively specific. The maximum reaction on second injection is always obtained when the substance which has sensitized is used, but in certain combinations intoxication can be produced by the other two substances. This intoxication, by a heterologous protein is "partial" and does not occur if the "complete" intoxication, produced by the homologous protein, has been effected; when "partial" intoxication has been produced by one or both of the heterologous substances, "complete" intoxication may still be effected by the homologous substance. The intensity of an homologous intoxication, after anaphylaxis by a single substance, would seem to vary somewhat with the substance used, the order of toxicity ranging, egg white, serum and last of all milk. After combined anaphylaxis, produced by initial injection of all three substances, the first intoxication, allowing of course a proper incubation period, may be produced by any one of the substances in question. When intoxications are effected with each substance in turn the serial set of symptoms varies according to the order in which the substances are injected on the subsequent days. When injected as the second or third of the series, egg white alone produces maximal symptoms at all times; horse serum is diminished in toxicity if used after either egg or milk and loses markedly if used after injection with both substances. Milk is very slightly toxic if given second in order and absolutely non-toxic if given third; this would compare with the actual toxic power of each substance as noted after homologous sensitization. The mixed anaphylaxis then is only relatively specific, since egg and horse serum will completely preëmpt the possibility of intoxication by milk if this substance is given last.

47 (303)

On the relation of calcium metabolism to tetany and the cure of tetany by administration of calcium.

By **W. G. MACCALLUM** and **CARL VOEGTLIN.**

[*From the Hunterian Laboratory, Johns Hopkins University.*]

Various researches, especially those of Jacques Loeb and J. B. MacCallum indicated a relationship between muscular twitching and the impoverishment of the tissues with respect to calcium. An endeavor was therefore made to determine whether the tetany produced by parathyroidectomy depended upon this condition. It was found that when the tetany was well developed it could be made to cease very rapidly by the intravenous or subcutaneous injection of a considerable dose of any soluble calcium salt, although the salts of other elements such as sodium or potassium had no such effect, but rather accentuated the symptoms. The analysis of the blood of dogs killed during tetany shows a calcium content about half that of the normal control dog and similarly the brain of the dog killed in tetany is poor in calcium as compared with that of the control dog, containing in fact only about half the normal amount. As far as the analyses are finished it appears that the output of calcium in the urine increases with the development of tetany.

From this it seems probable that the parathyroid glands exercise a control over the calcium metabolism so that when they are destroyed these processes do not go on normally but the tissues lose calcium and in this impoverished state become irritable, quiescence being restored by the reintroduction of considerable amounts of a soluble calcium salt.

It seems probable that the administration of calcium by injection or by the mouth may be useful in tiding over the severe symptoms in cases of tetany developing spontaneously or as the result of operation.

48 (304)

The relative toxicity of the chlorides of magnesium, calcium, potassium and sodium.

By **DON R. JOSEPH** and **S. J. MELTZER.**

[*From the Department of Physiology and Pharmacology of the Rockefeller Institute for Medical Research.*]

Sodium, potassium, calcium and magnesium are normal constituents of the animal body. However, when introduced intravenously in larger quantities they may have a poisonous effect. Pharmacologists frequently speak of the effect as a salt action. This means, at least according to the definition of some pharmacological writers, that the effect is due to osmotic changes. If this were true, the toxicity of all four inorganic, basic elements ought to be the same if introduced in the same molecular concentration. Although, as far as we know, a direct comparative study of the toxicity of these inorganic substances has never yet been made, a glance at the various data in the literature suffices to show that these substances differ greatly in their toxicity. As far as we can gather, it is generally assumed that potassium is the most poisonous; calcium seems to be considered a good deal less poisonous than potassium, but definitely more so than magnesium. As to sodium, only recently Loeb and his pupils called attention to its poisonous effect.

In a series of experiments on dogs, we compared the toxicity of these four inorganic elements in intravenous injections of their chlorides. In every instance molecular solutions were employed. The toxicity of each of these salts was tested on twelve animals. The injections were made by four different methods, using three dogs for each method, namely, by injections through the jugular vein, through the femoral vein, through the splenic vein, and intra-arterially, through the central end of the carotid artery. In the latter case the solution was driven by high pressure through a capillary tube. Except in the case of sodium chloride, the solutions ran in uniformly one c.c. per minute. In order to be able to finish an experiment in one day, the solutions of sodium chloride had to be run in at the rate of two c.c. per minute. Each of the solutions was permitted to run in until the animal died.

We shall give here no details, but state the results in the briefest possible way.

The average quantity of each salt per kilo which caused death, was as follows: $MgCl_2$, 2.35 c.c.; $CaCl_2$, 4 c.c.; KCl, 6.23 c.c., and NaCl, 63.24 c.c. These figures refer to the crystalline salts when dissolved in molecular solution. When, however, these values are reduced to that of the anhydrous salts, the figures read as follows: The fatal dose of magnesium chloride is 0.223 gram per kilo (dog); of calcium chloride it is 0.444; of potassium chloride 0.464; and sodium chloride is fatal only when 3.7 grams of the salt are given per kilo. In other words, magnesium chloride is twice as toxic as calcium chloride or potassium chloride. Again, potassium chloride is about eight times more toxic than sodium chloride. In the case of the latter we have to remember that the solution ran in twice as rapidly as the solutions of the other salts, which means that the comparative toxicity of sodium chloride is even much less than appears in the above scale.

49 (305)

The action of calcium upon the pupil and its relation to the effects of mydriatics.

By **JOHN AUER** and **S. J. MELTZER.**

[*From the Department of Physiology and Pharmacology of the Rockefeller Institute for Medical Research.*]

The intravenous injection of calcium exerts a pronounced effect upon the mechanism of pupilary constriction. The solution employed was *m*/8 $CaCl_2$; this was injected slowly through the external jugular vein of rabbits; in some instances the ear veins were used. After 12 to 14 c.c. of the solution had run in, stimulation of the cervical sympathetic nerves no longer caused dilatation of the pupil. At the same time, the pupil appeared to be smaller than normal and reacted less readily to light. When 20 to 25 c.c. had entered, the pupils, as a rule, became almost pinpoint in size. If the infusion was now stopped, the pupils remained contracted for about thirty minutes, and usually about two hours elapsed before the pupils again reached their normal size. There

were some exceptions in which a larger dose had to be infused before the pupil showed a distinct reaction to the calcium, and then the contraction did not become maximal. These exceptions seemed to occur when the rabbits were under profound ether anesthesia.

Atropin did not prevent this calcium myosis, but it retarded somewhat the onset of this myosis and hastened its disappearance after stoppage of the calcium infusion. Instillations of atropin into the conjunctival sac were a little more effective than intravenous injections. As far as we know calcium is the only substance which is able to overcome the full mydriatic effect of atropin.

What was said of atropin holds good, in general, also for cocaine; calcium overcomes the mydriatic effect of cocaine, but to a less extent than that of atropin. The mydriatic effect of the cocaine becomes especially active during the onset and the later period of the calcium myosis.

Ether antagonizes moderately the calcium myosis. This is especially well seen when a few c.c. of ether are injected subcutaneously after a calcium infusion. The contracted pupils of the rabbit now dilate fairly rapidly. As already mentioned, the calcium myosis develops less readily when the animal is deeply under ether.

The action of adrenalin in animals whose superior cervical sympathetic ganglion has been removed, is interesting. In these rabbits, as is now well known, the instillation or subcutaneous, intramuscular, or intravenous injection of adrenalin causes a strong, long lasting dilatation of the pupil on that side where the ganglion was removed. Calcium overcomes this dilatation also, but the mydriatic adrenalin-effect appears in a striking manner when the calcium infusion is discontinued. While the pupil on the side with intact ganglion remains strongly contracted, the pupil on the operated side becomes very wide and remains so for hours.

We have finally to mention that asphyxia also opposes moderately the myosis brought on by calcium.

We shall not enter here into a discussion of the intricate problem involved in the mechanism of calcium myosis; our present assumption is that this calcium myosis is produced by a stimulation and contraction of the muscle fibers in the pupilary sphincter.

50 (306)

The destruction of strophanthin in the animal organism.

By **ROBERT A. HATCHER** and **HAROLD C. BAILEY.**

[*From the Laboratory of Pharmacology of Cornell University Medical College.*]

Strophanthin is toxic in much smaller doses when injected into a vein or subcutaneously than when given by the mouth.

Dilute solutions injected slowly or at intervals are approximately as toxic as stronger solutions injected at once.

Strophanthin is not readily destroyed by the liver; no difference could be observed in the amounts required to cause death from slow injections of dilute solutions into the superior mesenteric, and into the femoral vein.

Peptic and activated pancreatic digests destroy only small amounts of the poison, not enough to account for more than a small part of the difference in toxicity by the mouth. Bile exerts little influence.

Ten times that amount of strophanthin which is fatal by the vein may disappear from the dog's intestine in an hour and a half without proving fatal, the portion remaining in the lumen of the intestine retains its toxicity, but death may result promptly (in thirty-eight minutes) from the introduction into the duodenum of an amount but slightly exceeding that necessary to cause death after oral administration.

The poison may be destroyed in part during its passage through the walls of the intestine, but since part of even a very small dose is absorbed unchanged, it seems fair to conclude that certain cells alone are concerned in the destruction while others permit it to pass through.

The cat and dog react to similar doses, the rabbit will stand somewhat larger doses administered subcutaneously and vastly larger doses by the mouth; this suggests that histological differences may furnish a clue to the cells concerned in the destruction.

One milligram of an active strophanthin given by mouth, per kg. of cat, has proved fatal, while a man has been given about as much (one hundred and fifty milligrams total) on two days.

The toxicity of strophanthin by the mouth can be varied at will to a considerable degree for the cat.

51 (307)

On the nature of the socalled glycogenolytic fibers in the greater splanchnic nerves.

By **J. J. R. MACLEOD.**

[*From the Physiological Laboratory, Western Reserve University.*]

The hyperglycæmia which invariably results from interference with pulmonic ventilation, produced either by constriction of the air passages or by inadequacy of the respiratory movements, has led to the question whether or not the hyperglycæmia following stimulation of the great splanchnic nerves may not also be due to an asphyxial condition induced locally in the liver. Such a local asphyxia of the hepatic lobule might be the result of diminished blood supply caused by constriction of the blood vessels in Glisson's capsule, or by the diminution of portal blood supply following constriction of the splanchnic vessels.

The question, therefore, presents itself as to whether stimulation of the great splanchnic nerve causes hyperglycogenolysis because secretory nerve fibers influencing the production or activity of the glycogenolytic ferment in the liver are contained therein, or because of a local disturbance in the blood supply of the liver following stimulation of vaso-constrictor fibers.

In the following communication a preliminary report is offered of several experiments devised to throw some light on these problems.

1. All the tissues running to the hilus of the liver except the portal vein were cut between peripherally and centrally placed ligatures. As much as possible of the outer coat of the portal vein was also removed. By these three operations all the hepatic nerves running from the cœliac plexus to the liver were severed. Stimulation of the great splanchnic nerve was found to cause no increase in the sugar content of the blood although the usual marked vaso-constriction of the splanchnic vessels occurred.

2. Clamping the portal vein for periods of about a minute at

intervals of about two minutes, produces only in some cases an increase in the blood sugar.

3. Ligation of all branches of the hepatic artery running to the liver produces no change in the sugar content of the blood.

4. Stimulation of the tissues adjacent to the portal vein — after doubly ligating and cutting — causes hyperglycemia in about fifty per cent. of the cases. Such stimulation does not usually cause any change in arterial blood pressure.

As a result of these four groups of experiments it would appear that local asphyxia of the hepatic lobule consequent upon changes in blood supply is a less likely explanation of hyperglycogenolysis than is the hypothesis which assumes the presence of glycogenolytic secretory fibers in the great splanchnic nerves.

If such fibers control the production of glycogenolytic ferment by the liver, we might expect atropin to paralyze the fibers. My observations in this direction, however, show that stimulation of the N. splanchnicus in atropinized dogs causes the usual hyperglycemia. Atropin itself, however, produces a hyperglycemia which makes the observation of doubtful value.

Experiments are in progress to determine by the method of Bang, Lyungdahl and Bohm[1] whether there is an increased amount of glycogenolytic ferment in the liver after stimulation of the great splanchnic nerve.

52 (308)

Prevention of syphilis in Macacus Rhesus by atoxyl.

By **SIMON FLEXNER.**

[*From the Rockefeller Institute for Medical Research.*]

The drug atoxyl has been employed successfully in causing the rapid disappearance of syphilitic lesions in human beings and in preventing the development of the specific inoculation eye lesions in rabbits. Metchnikoff reported recently that the same drug would prevent the development of the specific lesion in monkeys even if administered some days after the inoculation.

[1] *Beiträge zur chemischen Physiologie und Pathologie*, 1907, ix, p. 408; x, p. 1; x, p. 312.

The experiment which I wish to report was made upon five Macac monkeys. They were all inoculated over both eyebrows with scrapings from a syphilitic papule of the tongue which had been shown by the dark field illumination microscope to be rich in living *Spirocheta pallida*. Two of the monkeys remained as controls and the other three received 0.15 gram atoxyl subcutaneously, one day, eight days and fifteen days respectively after the inoculation. Only the two controls developed specific lesions. Three months later two of the atoxyl-treated monkeys (one having died in the interim) and another control monkey were inoculated over the right eyebrow with virus from a primary syphilitic lesion rich in *Spirocheta pallida*. Within three weeks one of the atoxyl-treated monkeys and the control monkey developed specific lesions, showing the pallida, over the right eyebrow.

This experiment is an example of the power which atoxyl possesses of suppressing the development of syphilitic lesions when given as late as fifteen days after inoculation with active virus, and indicates that this suppression is not attended with the production of a state of immunity to the virus.

53 (309)

Further notes on a rat tumor.

By **SIMON FLEXNER** and **J. W. JOBLING.**

[*From the Rockefeller Institute for Medical Research.*]

On several occasions we reported to this Society upon a rat tumor that has been transplanted for more than two years. The tumor was originally described as a sarcoma. In a recent report[1] we described its transformation into a malignant adenoma. This change in histological structure was attended with the acquisition of the property, hitherto absent, of producing metastasis in lymphatic glands. The reverse changes, namely from the adenoma into sarcoma, had been noted by Ehrlich, Leo Loeb and others in mouse cancers and were attributed to gradual or rapid proliferation and predominance of the stroma of the tumors or of a corresponding tissue derived from the host. Since our rat tumor

[1] This journal, 1908, v, p. 52.

underwent the change into adenoma when implanted beneath the skin, the epithelial cells must have arisen from elements present in the graft. The original tumor had been kept and sections were made from different parts of it in an endeavor to discover undoubted evidences of epithelial proliferation. Such evidences were found in several places, but notably in one place, in the glandular tubules or epithelium-lined spaces of the seminal vesicle in which organ the tumor developed originally. Hence there is no longer any doubt of the existence of a carcinomatous element in the original growth although it was restrained by the other and less highly organized parts of the tumor. The carcinomatous elements gradually gained supremacy in one strain of the tumor, then in other strains, until now all the strains which have been kept alive have either gone over into adenoma or are well advanced in this transformation.

54 (310)

On nucleic acids.

By **JOHN A. MANDEL, W. A. JACOBS** and **P. A. LEVENE.**

[*From the Rockefeller Institute for Medical Research.*]

In recent years, several substances have been obtained which resemble one another in the fact that all of them contain in their molecules phosphoric acid and a sugar, but which differ one from another in the number and in the character of the nitrogenous radicals contained in their molecules. To this group of substances belong: (1) glucophosphoric acid; (2) inosinic acid and guanylic acid; (3) yeast nucleic and triticonucleic acid; and (4) thymonucleic acid.

All these substances may be classified as nucleic acids:

1. The first substance is a glucophosphoric acid proper.

2. Inosinic and guanylic acids are monopurin-glucophosphoric acids. Each of them contains in its molecule only one purin base besides the glucophosphoric acid.

3. Yeast and triticonucleic acids each contains two purin and one pyrimidin radical in its molecule and may be regarded as di-purin-monopyrimidin-glycophosphoric acid.

4. Finally, thymonucleic acid is a dipurin-dipyrimidin-glucophosphoric acid.

Conclusions as to the nature and existence of the monopurin-glucophosphoric acid have passed through several phases during the past year. Last summer a paper by v. Fürth and Jerusalem appeared in which the existence of the substance was denied. However, within a short time, work done by Steudel, by Jones and by ourselves has not only established the existence of the substance, but also has shown that its occurrence is more general in animal organs than has hitherto been conceded. In fact, with the acceptance of this discovery, some investigators are inclined to regard thymonucleic acid as simply a mixture of different monopurin-glucophosphoric acids.

This view is not supported by our experiments. Both the carbohydrate and the purin bases are easily obtained on decomposition of guanylic acid, while great difficulty is experienced in obtaining these substances quantitatively, by hydrolysis of thymonucleic acids.

However, it is possible that all nucleic acids resemble one another in the order in which the components are linked together. There is support for the assumption that the carbohydrate is joined to the phosphoric acid and the base to the carbohydrate in a glucoside form. Thus, upon hydrolysis of inosinic acid by means of alkali, a condition may be obtained in which the original solution is not changed in its rotatory power and does not show reducing action on Fehling's solution, but which gives evidence of the presence of free phosphoric acid. On the other hand various workers report experiments in which on hydrolysis of inosinic acid, a glucophosphoric acid was obtained which had the power of reducing Fehling's solution and which formed phenylosazone.

Furthermore on partial hydrolysis of thymonucleic acids, substances are obtained which contain only purins or only pyrimidins linked to a carbohydrate. These substances have no reducing power, but on further cleavage yield levulinic acid.

These considerations, and also the results of our analysis of the bases lead us to believe that the accepted view of the elementary composition and of the structure of the thymonucleic acids has not yet been fully demonstrated.

It seems to us possible that thymonucleic acid consists not of a tetra- but of a penta-phosphoric acid. On this assumption and on further assumptions that the oxypurins and oxypyrimidins form anhydrids with the corresponding sugars, one would deduce the following formula for nucleic acid: $C_{51}H_{71}N_{20}O_{37}P_5$.

Calculated.	Found by Levene in 1905.
C 37.0 %	C 37.78 %
H 4.0	H 4.86
N 16.0	N 16.51
P 9.0	P 8.91

55 (311)

Regarding the innervation of the blood vessels of the kidney.

By **R. BURTON-OPITZ** and **DANIEL R. LUCAS.**

[*From the Physiological Laboratory of Columbia University, at the College of Physicians and Surgeons.*]

The blood flow through the left kidney was determined by means of the stromuhr described previously.[1] Having determined that the left splanchnicus major, as well as the plexus renalis, contains vaso-constrictory fibers for the corresponding kidney, different fibers of the plexus were isolated and stimulated separately. Of five nerve-fibers tested, four proved to be constrictory and one dilatory. The constricting fibers displayed different grades of effectiveness.

By cutting the fibers composing the plexus, a faster blood flow was obtained. Cutting the nervi vagi in the neck produced a slowing of the renal bloodflow. Division of these nerves above the diaphragm did not seem to change the flow very markedly.

In another series of experiments the right splanchnicus was stimulated while the bloodflow through the left kidney was being measured. The results indicate that the innervation of the kidney is bilateral. Although stimulation of the right splanchnic produced a vaso-constriction in the left kidney, the effect was much weaker and more gradual than when the left nerve was used.

[1] Burton-Opitz: This journal, 1907, iv, p. 24.

56 (312)

Regarding the innervation of the blood vessels of the intestine.

By **R. BURTON-OPITZ.**

[*From the Physiological Laboratory of Columbia University, at the College of Physicians and Surgeons.*]

Quantitative measurements of the blood flow in the mesenteric vein proved that the innervation of the blood vessels of the intestine by way of the splanchnic nerves is bilateral. Vasoconstrictory effects were produced on stimulation of the left and right splanchnicus major.

Cutting the cervical parts of the nervi vagi caused a marked slowing of the blood stream. Division of the vagi above the diaphragm produced a similar but much milder effect.

The experiments also tend to show that the vagi contain vasoconstrictory fibers for the intestine.

57 (313)

Note on anaphylaxis to horse serum.

By **PAUL A. LEWIS.**

[*From the Antitoxin Laboratory of the Massachusetts State Board of Health.*]

Young guinea pigs, bred from mothers which have been treated with a mixture of horse serum and diphtheria antitoxin, are found very susceptible to the toxic action of horse serum. Recently I have had the opportunity to study the blood of these guinea pigs hypersusceptible by reason of their descent and can now contrast its properties with those of the blood of the animals hyper-sensitized by direct treatment. The blood of the animals directly or actively sensitized contains a substance which when the blood is transferred to untreated young animals of normal descent, renders them immediately (within 24 hours) hypersensitive to horse serum. It also contains a substance which renders "fresh" animals to which it is transferred hypersensitive, after an incubation period corresponding to that required for direct sensitization by horse serum. This substance, designated as "anaphy-

lactin" by Gay and Southard, who discovered it, is much more potent than that which acts immediately if its power is stated in inverse terms of the amount of blood which must be transferred in order to develop the reactions. One tenth to one cubic centimeter will give results after two weeks while fifteen cubic centimenters are needed to develop the possibility of reaction after twenty four hours.

In the study of the animals hypersensitive by breeding, these distinctions become greatly emphasized. When the blood of such animals aged four or five weeks, is transferred in quantity (15 c.c.) to fresh guinea pigs they become sensitive to the toxic action of horse serum within twenty four hours. But whether the quantity of blood used be large or small, the anaphylactin or substance sensitizing after an incubation period, cannot be demonstrated.

These experiments were undertaken in the belief that the young born of serum-treated mothers were probably rendered hypersensitive by a passive process, analogous, although of opposite result, to the transfer of immunity from mother to offspring. Taken alone or in conjunction only with facts so far developed in regard to this reaction they support this view. Certain experiments not as yet concluded have shown, however, that the sensitiveness of the young animals may last longer than had been supposed. Certain other results, which on control are found to be clearly exceptional, point to the possibility of an influence extending from the treated mother to her grandchildren through the female line. In the hope of extending these observations I defer drawing definite conclusions on these points.

Some experiments on the reaction of the rabbit to horse serum, incomplete from the point of view with which they were begun, have shown that this animal is much more difficult to sensitize to the point where the intravenous injection of serum will cause sudden death, than is the guinea pig. The toxin-antitoxin mixture so effectual for the latter animal does not sensitize the rabbit at all. It would therefore seem most unwise at present to draw conclusions from the recent work on anaphylaxis in animals which would influence in any way the therapeutic use of specific sera.

58 (314)

A study of "protagon" prepared by the Wilson-Cramer method.

By **L. J. Cohen** and **William J. Gies.**

[*From the Laboratory of Biological Chemistry of Columbia University, at the College of Physicians and Surgeons.*]

Two months ago we received a copy of the January issue of the journal containing Wilson and Cramer's paper on "Protagon."[1] Results recently published from this laboratory[2] and also from Halliburton's,[3] appeared to prove conclusively that protagon is always merely a mixture of substances and that it has no constant chemical qualities because of its invariable mechanical heterogeneity. Wilson and Cramer view our data from another standpoint, however. They have presented a few results which they believe completely invalidate our prior conclusions against the chemical individuality of protagon. We cannot agree with them in this opinion and are surprised to find their paper so weak in support of the far-reaching deductions they summarize on its concluding page.

Numerous inconsistencies in the paper by Wilson and Cramer must be frankly considered if the truth regarding protagon shall prevail. In this preliminary communication, however, we shall refer chiefly to the revival of the old and frequently abandoned notion[4] that treatment of protagon with warm alcohol effects its *chemical decomposition* which, they say (p. 105), "has not been suspected previously."

Regarding the action of warm alcohol on protagon, Wilson and Cramer say: "The statement made in a former paper by Cramer, that protagon is not decomposed by warm ether or boiling alcohol, must therefore be corrected. *In the case of boiling alcohol it is true only if the solvent is prevented from acting on*

[1] Wilson and Cramer: *Journal of Experimental Physiology*, 1908, i, p. 97.

[2] Lesem and Gies: *American Journal of Physiology*, 1902, viii, p. 183; Gies and collaborators: Biochemical Researches, 1903, i, p. 263 (reprint no. 11); Posner and Gies: *Journal of Biological Chemistry*, 1905, i, p. 59; Gies: The same, 1906, ii, p. 350; Steel and Gies: *American Journal of Physiology*, 1907, xx, p. 378.

[3] Rosenheim and Tebb: *Journal of Physiology*, 1907, xxxvi, p. 1.

[4] Gies: *Journal of Biological Chemistry*, 1907, iii, p. 350.

protagon for some time."[1] Wilson and Cramer attribute the hypothetical decomposing effect of warm alcohol on protagon to an assumed "hydrolyzing (!) action." They present no facts, however, that justify this guess.

Wilson and Cramer have prepared protagon by a new method, in which the brain tissue is extracted first with cold 96 per cent. alcohol, later with cold ether, for the complete removal of cholesterin and lecithin. The brain mass remaining after this treatment is then pulverized and extracted with boiling absolute alcohol. "The boiling solvent is poured on the powder and the mixture *kept boiling for one or two minutes in a warm bath, moving the mixture all the time.*[2] The alcoholic solution is filtered through a hot-water funnel; the filtrate is allowed to drop into a vessel cooled in ice. The same process of extraction is repeated twice. The crude crystalline product is washed with ether and dried in vacuo. Recrystallization is effected by pouring boiling absolute alcohol on the sample of protagon. The solution is kept boiling for one minute and then filtered as before."

The protagon products prepared by Wilson and Cramer by this method contained between 0.9 per cent. and 1.0 per cent. of phosphorus, after the fourth or fifth recrystallization. The general composition of their preparations is about the same as that of the products previously made by Cramer[3] by a method which, they assume, as quoted above, involved decomposition and *the ultimate isolation, therefore, of a mixture.*

Wilson and Cramer refer with great satisfaction to the "constancy of the chemical composition" of their products as something very striking and important, but, as they fail to give such details of their treatment as the mutual proportions of the protagon and the solvent employed in their recrystallizations, we are not at all impressed by the analytic constancy referred to.

As was stated above, Wilson and Cramer believe that Gies and his collaborators unknowingly subjected their protagons to *decomposition* by the "hydrolyzing action" of the warm alcohol used in the fractional recrystallization experiments that yielded the re-

[1] Italics our own.

[2] Italics our own.

[3] Cramer: *Journal of Physiology*, 1904, xxxi, p. 31.

sults which have lately made protagon appear to be a mechanical mixture. It is very noticeable, however, that Wilson and Cramer, in spite of their insistence upon this conclusion, have failed to give their opinion a thorough test. They have not ascertained whether fractional recrystallization of protagon by their own non-decomposition method does not yield results similar to those they ascribe to hydrolytic decomposition in the experiments in this laboratory. They declared that treatment with boiling absolute alcohol, for from one to two minutes at a time by their new method, does not bring about such hydrolytic decomposition. Then why did they not show that fractional recrystallization, by their non-decomposition method, yields fractions of identical composition?

We have endeavored to settle the doubt in this matter on the ground selected by Wilson and Cramer. We have prepared protagon by carefully following every detail of their very simple method, as given by them. We cannot believe that defects in our technique have accounted for any of our results. Nevertheless, we obtained protagon which *steadily decreased in phosphorus content each time it was recrystallized by the Wilson and Cramer method* (1.3 per cent. to 0.4 per cent. in four recrystallizations); which never completely redissolved in the moderate excess of boiling absolute alcohol we used for the purpose; which, on recrystallization, each time left in the mother liquor material having a much higher phosphorus content than the corresponding crystallized portion; and which, on fractional division by recrystallization at 20°C. and 0°C., yielded two crystalline products, and material in the final mother liquor, that were *strikingly unlike, physically and chemically*. In short, simple fractional recrystallization of protagon made by the Wilson-Cramer method, in which treatment, according to Wilson and Cramer, *decomposition does not occur*, has given results which confirm conclusively the data and deductions published by Gies and his collaborators, that Wilson and Cramer have sought unsuccessfully to explain away.

Rosenheim and Tebb[1] also recently examined Wilson and Cramer's results, but have not been able to confirm Wilson and Cramer's conclusions.

[1] Rosenheim and Tebb: Proceedings of the Physiological Society, *Journal of Physiology*, 1908, xxxvii.

In a more detailed publication of our results we shall shortly draw attention to the fact that if certain of the statements made by Wilson and Cramer are correct, Gamgee's protagon cannot be regarded as a definite chemical compound, but must be accepted as a mixture, quite unlike the product that Wilson and Cramer now designate as protagon. Wilson and Cramer consider Gamgee's protagon identical with their own and the standard product for all protagon comparisons. They also appear to believe that anything having the quantitative elementary composition of Gamgee's protagon, whatever the method employed to prepare it, is protagon.

Twenty ninth meeting.

Cornell University Medical College, New York City. May 20, 1908. Silas P. Beebe in the chair.

59 (315)

Heredity of some human physical characteristics.

By **C. B. DAVENPORT.**

[*From the Carnegie Institution of Washington; Station for Experimental Evolution. Cold Spring Harbor, Long Island, N. Y.*]

It has already been shown[1] that dark brown eye color is a Mendelian dominant to gray and gray to blue. It now appears that the form of the hair also follows Mendel's law. Thus, if the three grades of straight, wavy and curly be recognized then straight hair is recessive to curly. But "wavy" is peculiar in that it appears to be a hybrid or heterozygous condition indicating the presence of both straight and curly germ cells. It follows from the foregoing facts that two blue eyed, straight haired parents will have only children like themselves; but two brown eyed, curly haired parents may have a variety of these characters in their families. Again, the records collected by Mrs. Davenport and myself show that two flaxen haired parents have flaxen haired children and probably only such; two parents with light brown hair have children with hair of that color and lighter, but not darker; while children of two parents each with dark brown or black hair produce children with all of the varieties of hair color. Also two golden haired parents have only golden haired children and none with red hair. Consequently two blue eyed, flaxen or golden and straight haired parents will have only children like themselves. And the reason is that the germ cells as well as the somatic cells of such parents lack the dark and curly characters.

In the foregoing cases of heredity the more advanced condition is dominant over the less advanced. This is often, if not usually, true and this rule enables us to predict the outcome of

[1] Gertrude C. and Charles B. Davenport, *Science*, November 1, 1907.

untried matings between parents having dissimilar characteristics. The more highly developed condition dominates over the less highly developed; and in the extreme case the presence of a character dominates over its entire absence. This extreme case gives results that accord with Mendel's law; the cases in which the characteristics of the parents differ merely in degree are often not inherited precisely in accordance with that law.

60 (316)

The experimental production of the maternal part of the placenta in the rabbit.[1]

By **LEO LOEB.**

[*From the Laboratory of Experimental Pathology, University of Pennsylvania.*]

In a former communication[2] I stated that I had been able to produce at will, decidual tumors in the uterus of the guinea-pig by making deep incisions into the wall of the uterus, at certain periods of the sexual cycle. The nodules thus produced belong to that class of formations which I designated as "transitory tumors." Since then, further investigations[3] have demonstrated that these deciduomata are produced under the influence of an internal secretion of the ovaries. There are indications that it is the corpus luteum which produces this internal secretion. If such a "preparing substance" has been secreted by the ovaries, indifferent stimuli are sufficient to call forth the development of the deciduomata. In the guinea-pig the new formations show a structure identical with that of the decidua. Large cells of connective tissue origin show an epithelial arrangement. The nuclei of many cells are hypertrophic, the cytoplasm is, on the whole, solid, and stains well with eosin. The decidual tissue of the placenta of the rabbit differs in some important respects from that of the guinea-pig, if we compare the placentas at the corresponding stages of development. In the rabbit the decidual cells are quite vacuolar and the

[1] This investigation was carried out under a grant from the Rockefeller Institute for Medical Research.

[2] Loeb: This journal, 1907, iv, p. 93.

[3] A report on these further investigations appeared in the Journal of the American Medical Association, June, 1908.

blood vessels show an interesting change. At a very early period the endothelial lining becomes transformed into syncytial and plasmodial masses. It was of interest to determine whether the deciduæ produced experimentally in the guinea-pig and in the rabbit differed in the same way as the normal deciduæ in these two species.

The following experiments were, therefore, carried out:

1. In four female rabbits which had been kept separated from males for some time previous to the operation and which were not pregnant, no deciduomata were formed after the usual incisions into the uterus had been made. We found only edematous mucosa on microscopic examination.

2. Three rabbits were operated, 3 to 5 days after copulation. In two of these animals the nodules, formed ten days after the operation, were examined microscopically. They showed the above mentioned characters of the maternal part of the placenta of the rabbit.

3. These latter experiments were, perhaps, open to the objection that ova had come into contact with that part of the mucosa which gave rise to the formation of decidual tissue. Although such an occurrence was extremely unlikely inasmuch as the continuity of the uterine cavity had been interrupted, through the operation, at many places, two other experiments were performed, in which, in each case, both fallopian tubes were tied at their junction with the uterine horns about 1¾ hours after copulation. Seven days later the ordinary incisions were made into the uterus. Ten days later many decidual nodules were formed in both cases, which, on microscopic examination, showed the characteristic features of the rabbit placenta, namely, the vacuolated cells, the plasmodial changes of the blood vessels, and besides, the formation of multinucleated cells in the uterine epithelial layer, which represented the first step in the formation of epithelial plasmodia.

These changes, however, did not occur at every point simultaneously. The plasmodial transformation of the blood vessels was sometimes seen without the accompanying formation of vacuolar cells; at other places both changes were present in the same area. On the whole, the nodules in the rabbit were, in proportion to the size of the rabbit uterus, considerably smaller than the deciduomata formed in the guinea-pig.

From these investigations the conclusion can be drawn that the experimentally produced deciduomata differ in the rabbit and in the guinea-pig in the same way as the natural placentas do. Whether the dissimilar reactions of homologous tissues to similar stimuli in the rabbit and in the guinea-pig depend upon a difference in the structure of the protoplasm, or upon a quantitative or qualitative difference of the internal secretion of the ovaries, or upon a combination of these two factors, are questions which cannot be answered at present.

61 (317)

Hemolytic action of the venom of Heloderma suspectum.

By **ELIZABETH COOKE** and **LEO LOEB.**

[*From the Laboratory of Experimental Pathology, University of Pennsylvania.*][1]

1. The venom of *Heloderma suspectum* alone does not cause hemolysis of blood corpuscles (ox, sheep, dog, guinea-pig, rabbit, *Heloderma*, frog). Fresh and dried venom behave alike in this and other respects. It must be mentioned that on a few occasions guinea-pig corpuscles showed a trace of hemolysis with venom alone, but as this usually did not occur, it is likely that the corpuscles which behaved as exceptions, had, in some way, lost part of their natural resistance.

2. In combination with lecithin the venom causes hemolysis. The amount of lecithin necessary varies for corpuscles of different species.

3. Certain blood sera, heated or unheated, act like lecithin (dog, horse, turtle), others do not (guinea-pig, rabbit, ox, sheep, *Heloderma*). Only those activate which are supposed to act through lecithin, not those which act through complement.

4. A number of non-activating sera, heated or unheated, inhibit the venom-lecithin hemolysis. Different sera possess different inhibiting values, the greatest observed inhibition being due to *Heloderma* serum and the next greatest to guinea-pig serum. But the

[1] These experiments represent part of an investigation upon the toxic action of venom of *Heloderma* carried out in this laboratory under a grant from the Carnegie Institution.

inhibiting action of a certain serum varies to some extent for different corpuscles. Details concerning these differences will be published later.

5. *Heloderma* serum does not activate its own venom.

6. Although *Heloderma* is naturally immune against the chief toxic effects of its own venom, the venom will, when added to lecithin or to an activating serum, cause hemolysis of the blood corpuscles of *Heloderma, in vitro.*

7. The hemolysin passes through a Berkefeld filter, dialyzes very slowly, resists a temperature of 100° C. for 10 minutes, and is wholly or partially destroyed by heating to 100° C. for 30 minutes. It is, therefore, relatively very resistant to heat.

8. As the preceding statements indicate, the hemolysin of *Heloderma suspectum* differs in several respects from the hemolysins of snake venoms. It may also be added that compared with such powerfully hemolytic agents as the venom of cobra, the venom of *Heloderma* is only weakly hemolytic.

62 (318)

The biological relations of seed proteins.

By **THOMAS B. OSBORNE.**

[*From the Connecticut Agricultural Experiment Station, New Haven, Conn.*]

At a recent meeting of this society, Reichert and Brown gave the results of their examination of the crystal forms of hemoglobins obtained from a large number of different species of animals.[1]

The crystallographic method was adopted because they believed that by this means they might succeed where the chemist had failed.

That the chemist has failed to establish differences between homologous proteins which are quite as marked as those indicated by the method employed by Reichert and Brown, I cannot admit.

Without wishing in any way to detract from the importance of the highly interesting results reported by Reichert and Brown, I would like to call your attention to the results obtained by purely chemical means in the investigations of the vegetable proteins

[1] Reichert and Brown: This journal, 1908, v, p. 66.

which have been made in my laboratory during the past years, and which are in complete harmony with the results of Reichert and Brown.

The most marked instance of the agreement of both physical and chemical characteristics of the protein constituents of any group of seeds with the biological relations of the seeds is shown by the proteins of the cereals.

The seeds of the cereals are the only ones containing protein soluble in relatively strong alcohol. These proteins yield no lysine, much proline and ammonia, relatively little arginine and histidine, and, with the exception of that from the maize — which, however, yields nearly 20 per cent. — very large amounts of glutaminic acid. There is likewise a close resemblance between these seeds in the character and proportion of the several different forms of protein which they contain.

The proteins of the leguminous seeds are very much alike but very different from those of the cereals. The proteins of the pea, horse bean, and lentil appear to differ from each other only in the proportion of their different forms, and from those of the vetch only in the absence of vicilin which is found in the other seeds.

The proteins of these four legumes differ distinctly from those of *Phaseolus* in properties, ultimate composition, and in the proportion of their decomposition products. Distinct differences have not been observed between the proteins of *Phaseolus vulgaris* and *Phaseolus radiatus*.

The proteins of the cow pea and the soy bean are different from one another, as well as from those of the legumes just mentioned, yet the proteins of all these seeds are very similar in most respects. The proteins of the lupines show the greatest divergence from the general type of leguminous proteins, but in the proportion of their decomposition products they are similar. There are differences between the proteins of *Lupinus luteus* and *Lupinus angustifolia*, although in most respects they are very much alike.

The proteins of such other seeds as have been carefully studied cannot be compared in respect to their botanical relations, because the seeds from which they have been obtained are not very nearly related to each other.

The results of my work have shown that no two seeds are alike in their protein constituents, and that those proteins which appear to be alike are found only in seeds that are botanically closely related.

As I have elsewhere pointed out, it would seem that these differences in the reserve food substances of the endosperm must have an important bearing on the character of the developing embryo which derives its first food from them.

This food substance, and the embryo as well, are the final products of the series of chemical changes which led to their formation. When the embryo begins its development it finds at hand a definite food; which for each individual of the same species is the same, but for the individuals of different species is different. Each member of a species begins its independent life under similar chemical conditions, but under chemical conditions which are different from those of every other species.

When, therefore, each individual plant reaches that stage of development at which its organs of assimilation are able to furnish it with nutriment from its external surroundings, it is highly probable that its chemical processes have already been established along definite lines which it must follow throughout the rest of its life.

63 (319)

Variation in hydrochloric acid secretion during the digestive period.

NELLIS B. FOSTER and ADRIAN V. S. LAMBERT.

[*From the Laboratory of Biological Chemistry of Columbia University, at the College of Physicians and Surgeons.*]

These observations were conducted on dogs having Pawlow double stomachs. After test meals of hashed meat, alone or mixed with crackers and water, it was noted that the percentage of HCl in the gastric secretion varied somewhat during the digestive period. Estimations of the HCl were repeatedly made for each hour during the period, by the Volhard method of chlorine determination, after neutralization of the juice with NaOH and careful incineration. The total chlorides and the metallic chlorides were determined, and the difference between them reckoned as HCl. By

the use of this method it became evident that the gastric secretion was higher in its content of HCl early in digestion than it was toward the end and that there was a general lowering of the acid percentage as digestion progressed, in the same way that there was a lessening in the amount of juice secreted. The amount of HCl in gastric juice in the first two hours of digestion was from 0.4 to 0.5 per cent., whereas in the last hour it sometimes fell to 0.2 per cent. or slightly lower.

The acidity of the juice and to some extent the variation in secretion varies with different dogs and with different diets, but with any one animal the response elicited by a certain test meal is remarkably constant, and the curves of acid secretion, like the curves for the amounts of juice secreted, are uniform.

64 (320)

The effects of some organic acids on the secretion of gastric juice.

NELLIS B. FOSTER and **ADRIAN V. S. LAMBERT.**

[*From the Laboratory of Biological Chemistry of Columbia University, at the College of Physicians and Surgeons.*]

The acids used in these experiments were given in food to dogs with Pawlow double stomachs. The following acids were employed in concentrations of 0.5 per cent. and 0.2 per cent. in all experiments: lactic, butyric, citric, acetic. Lemon juice (7 per cent. citric) and vinegar (4.8 per cent. acetic) were also used. In no case, with the possible exception of vinegar, could a stimulating action on the gastric glands be attributed to the acid liquids administered. The juice secreted, in amount and acid content, was uniform with the controls. In some cases vinegar appeared to excite a more copious secretion of juice but this result was not constant. The distillate from neutralized vinegar acted in the same way as vinegar, while acetic acid alone appeared to be inert. From this it may be inferred that if vinegar has any effect upon digestion, it is due to the volatile substances which it contains and not to the acetic acid.

65 (321)

The effect of mechanical obstruction of the pyloric outlet on gastric secretion.

By **NELLIS B. FOSTER** and **ADRIAN V. S. LAMBERT.**

[*From the Laboratory of Biological Chemistry of Columbia University, at the College of Physicians and Surgeons.*]

Obstruction at the pylorus was induced in dogs with Pawlow double stomachs, by passing a silver band around the pylorus and regulating the constriction so as to narrow but not entirely occlude the pyloric lumen. The effects of this operation upon gastric secretion were primarily: (1) A diminution in the amount of gastric juice secreted in the first two hours after taking food, that is, there was a decrease in the amount of appetite juice. (2) The digestive period was much prolonged, a copious secretion continuing into the seventh and eighth hours, when, under normal conditions with the same foods, no secretion took place after the fourth hour. (3) There was a constant secretion of gastric juice without regard to the time of the last feeding and having no apparent relation to whether the stomach contained food or was empty, and also (4) a marked hypertrophy of the muscle coats of the stomach at the pyloric end, with moderate dilatation of the cardiac portion.

66 (322)

Transplantation of devitalized arterial segments.

By **ISAAC LEVIN** and **JOHN H. LARKIN.**

[*From the Department of Pathology of Columbia University, at the College of Physicians and Surgeons.*]

Before entering upon the discussion of the subject matter, we will take this opportunity to pay our tribute to the pioneer in the surgery of blood vessels. On the eighteenth of last April, Dr. N. V. Eck died in St. Petersburg. Nearly a decade ago he succeeded in uniting the portal vein to the cava in a dog, thus performing the operation for the Eck fistula.

Carrel demonstrated that it is not only possible to unite the two ends of a severed artery by a circular suture, but also to interpose between the cut ends a segment of an artery of another

animal and perform a double anastomosis. The success of the operation is due to the fine technique elaborated by Carrel.

Now, the question arises, upon what does this success depend? This question has not only a technical, operative, but a general physiological interest. To implant an arterial segment successfully means to avoid an intravascular thrombus after the operation.

According to the generally accepted theory of Brücke, blood remains fluid as long as it circulates in a vessel lined with an unimpaired living endothelium. Should the endothelium be injured through infection or trauma, a thrombus must form. In Carrel's experiments with implantation of arterial segments of the same species of animals, it seemed probable that the implanted segments remained alive. But in implantation of arterial segments of different animal species, the question presented itself whether these segments really remain alive, or, whether it is possible to implant *devitalized* arterial segments and the theories of intravascular coagulation of blood have to be revised. Our experiments consisted in implantations of a segment of an aorta from a dog, about one inch long, hardened in 4 per cent. formalin, into the abdominal aorta of another dog.

On January 23, 1908, we performed the first experiment. For ten days the dog did well; there was normal pulsation in both femoral arteries. On the eleventh day, the animal was found in the morning with protruding intestines. A secondary laparotomy was performed, but the dog died during the day. Both anastomoses held perfectly; the implanted piece was patent, without any thrombi. Microscopically there was hardly any cellular structure to be found in the implanted piece, but the elastic fibers could be seen in nearly normal quantity.

We then did some unsuccessful experiments on implantation of boiled arteries and ureters (from a freshly autopsied human body) hardened in formalin, in a dog and cat.

Meanwhile, Guthrie reported successful implantation of formalized segments into the carotid of a dog. We repeated the experiments and must state that in places where we felt pulsation during life the implanted segment contained an organized thrombus with numerous blood vessels running through it.

On May 8, last, we performed another experiment with implan-

tation of a formalized segment in the abdominal aorta of a dog. The animal had normal pulsation in the femoral arteries and was otherwise doing well with the exception of a subcutaneous infection of the abdominal wound. On May 18, we gave the animal anesthesia in order to dress the wound, and it died in a few hours apparently from the influence of ether.

The specimen of the aorta presents a very interesting condition. The distal part of the segment is free from thrombus, the anastomosis is perfect, in the proximal portion there is a large thin parietal thrombus covering the suture line, and also a small gangrenous part of the segment.

Our research is not yet concluded, but the results obtained present some points of theoretical importance.

For ten days the circulating blood was passing through a dead canal over one inch long, and it remained fluid. It seems, then, that blood need not necessarily run through a vessel lined with an unimpaired endothelium in order to remain fluid.

The implantation of devitalized segments is technically a great deal more difficult than that of living ones. If the implanted segment is of the same size as the rest of the artery then the anastomosis is easy to perform but as soon as the clamps are removed both the afferent and the efferent parts of the artery become wider than the implanted segment and the result is thrombosis. In order that the size of the segment should correspond after implantation to the rest of the artery, we have to use a segment wider than the rest of the artery and this increases the technical difficulty.

67 (323)

A study of nitrogen metabolism in a case presenting short paroxysms of fever of unknown origin.

By **THEODORE C. JANEWAY** and **HERMAN O. MOSENTHAL**

[*From the Laboratory of Biological Chemistry of Columbia University, at the College of Physicians and Surgeons.*]

The patient upon whom these studies were made presented an unusual paroxysmal syndrome probably toxemic in origin, characterized by attacks of prostration, pain — chiefly abdominal — fever, tachycardia and polynuclear leucocytosis, with some nausea and

vomiting, lasting twenty four to forty eight hours. The case was reported by us to the Association of American Physicians, May 12. A study of the urinary nitrogen was made November 6 to December 2, '07, and April 7 to May 5, '08, embracing three attacks during the first period and two during the second. Urea and ammonia nitrogen did not vary beyond normal limits. The chief interest centered in the uric acid and creatinin output. During the latter part of the first period the uric acid, on a uniform purin-free diet of 2,350 calories for a girl of 47.3 kilos, showed a distinct fall before the last attack, with a subsequent rise. During the second period the patient was kept on a creatin and purin-free diet unrestricted as to quantity, the amounts of creatinin and uric acid rising sharply after each attack, the latter showing a fall before the attack. Throughout the whole period the uric acid showed as wide variations as Kaufman and Mohr, and von Noorden and Schliep, described in the subjects of true gout. A test of her tolerance for exogenous purins, however, showed that her elimination of uric acid after eating 580 grams of beef was even higher than Burian and Schur's normal figure of 50 per cent. The creatinin was also wholly eliminated. Comparison of the eliminations during these two days with those in the urine after the attacks shows that, in the latter urines, she excreted as much additional uric acid and creatinin as might be derived from 580 grams of beef, with a nitrogen loss of 10 grams after the first attack and 13 grams after the second. The increased nitrogen output on the two meat days over the previous average was about 9 grams. The increase in the uric acid and creatinin, therefore, would seem to correspond closely with the amount of toxic tissue destruction that occurred in these short paroxysms of fever.

68 (324)

Histological changes in transplanted blood vessels.

By **WILBUR WARD.**

Communicated by Francis Carter Wood.

[*From the Department of Clinical Pathology of Columbia University, at the College of Physicians and Surgeons.*]

It has been shown by many experimenters that segments of blood vessels may be transplanted to other vessels, in animals

either of the same or of different species, and continue, for a certain period of time at least, to fulfil all physiological requirements. Various changes may be noted in the gross appearances of the transplanted segments, depending on the vessel transplanted, the site of transplantation, and the animal into which the implantation takes place, whether of the same or different species, closely or distantly related.

A segment of a rabbit's aorta was placed in the carotid of a dog, by the Carrel method of suture, and removed at the end of seventy days. The mechanical function of the vessel was perfect. The lumen was moderately dilated, and the walls slightly thinned. Upon microscopical examination, however, the normal structure of the transplanted segment had almost entirely disappeared. The intima had disappeared, being replaced by a layer of hyalin fibrin and blood; the muscular elements had either disappeared or been greatly reduced in number, while the various layers had been the seat of many fine hemorrhages, which showed as small masses of hyalin fibrin into which new connective tissue had penetrated. New fibrous tissue had replaced the whole structure of the vessel wall to a marked degree. The striking change, however, was the absolute disappearance of all the elastic tissue in the transplanted segment. Sections stained by Weigert's elastic tissue stain showed no elastic tissue in the piece of rabbit's aorta, while that in the carotid of the canine host was normal and ran up to the line of suture, where it stopped abruptly.

The entire disappearance of elastic tissue has not been observed in segments transplanted from one animal to another of the same species. A segment of a dog's aorta implanted in the aorta of a second dog, and examined at the end of seventy days, shows a very slight and scarcely appreciable diminution in the amount of elastic tissue in the transplanted segment, the slight loss being in the finer fibrils. There is no regeneration of elastic tissue, as there is a well defined wedge of connective tissue, without elastic elements, between the sutured ends of the cut vessels. The muscular and connective tissue layers are well preserved; the endothelium is missing in some places.

The more widely separated the species of animals used, the more rapid and complete is the disappearance of the elastic tissue.

Sections of a cat's aorta placed in the carotid of a dog, showed at the end of twenty days a very slight loss of elastic tissue, with the beginning of a break down of the individual muscle fibers, and proliferation of new fibrous connective tissue.

These observations of the behavior of the elastic tissue add weight to the general proposition that the cells of vessels transplanted from one animal to another of a different species do not actually survive, but are gradually broken down and absorbed, this process usually being slow enough to allow of sufficient new fibrous connective tissue formation (probably by the tissues of the host) to permit the function of the vessel to be maintained for a considerable period of time at least. The ultimate fate of such transplanted segments can be determined only by more protracted experiments.

69 (325)

Note upon the supposed presence of a gastric hormon in the salivary glands.

By **A. S. LOEVENHART** and **D. R. HOOKER.**

[*From the Physiological Laboratory of Johns Hopkins University.*]

There was published in the Proceedings of this Society in June of last year [1] a statement of the results of clinical and experimental observations upon the influence of the salivary glands on the secretion of normal gastric juice. It was stated that in four cases of Mikulicz's disease, uncomplicated, no gastric juice was secreted during the disease. The experimentally analogous condition brought about by removal of all the salivary glands in dogs also gave the same result, namely, a permanent stoppage of the normal gastric secretion. It was stated that when food, insalivated by other healthy dogs, was given to such animals no flow of gastric juice followed. If, however, extracts made of salivary glands from normal dogs, were injected, either intravenously or intraperitoneally, a temporary resumption of gastric secretion occurred after, as well as before, section of all extrinsic nerves to the stomach. The author, as the result of these observations, concluded that the normal gastric secretion depends in part upon an internal secretion of the salivary glands.

[1] Hemmeter: This Journal, 1907, iv, p. 153.

In view of the importance both of the results and of the conclusions presented in this communication, any attempt at confirmation of them would seem worthy of brief notice. An opportunity to make this attempt was given us through the kindness of Dr. Cushing who furnished us with two dogs with simple gastric fistulæ.

It was therefore decided to make a few experiments with the idea of testing directly whether or not extracts of the salivary glands are capable of stimulating a flow of gastric juice. If Hemmeter's main contention be correct, it would be supposed that the gastric hormon obtained by extracting the salivary glands should produce a decided effect upon the secretion of gastric juice, when introduced directly into the blood of a living animal. It is to be noted that in our experiments extracts were made only of the submaxillary gland, and that these extracts were injected intravenously only into normal animals, not into animals with the salivary glands removed.

It may be stated at once that the results of these experiments were entirely negative, and, so far as they go, they fail to corroborate Hemmeter's view of the presence of a gastric hormon in the salivary glands. The details of the experiments are as follows:

Dog 1. *Operation.* Simple gastric fistula. Wound healed *per primum.*

Extract. Two submaxillary glands from a healthy dog which died during the first operation under morphin-ether anesthesia, the glands being removed immediately after death. Glands at once (5.30 P. M., Nov. 9) macerated with sand in 0.7 per cent. NaCl, boiled and filtered. Put sterile in ice-box. Again filtered and sterilized before injection.

November 11, 1907. Animal appeared to have distemper; feverish with slight discharge from eyes and nose. Was thin and janitor said had little appetite. Last food twelve hours previously. 3.45 P. M. Stomach catheterized and found empty. Dog allowed to run about room. 4.00. Catheterized. About 15 c.c. clear juice with a few fine floculi. 4.15. Catheter continuously in stomach. Dog quiet and contented. Ear vein exposed by slight skin incision. 4.30. About 15–20 c.c. salivary extract injected intravenously. No juice. 4.50. Dog allowed to smell cracker. Gush of a few drops of fluid from catheter coincident with act.

Flow did not continue. 5.00. Dog allowed to smell meat. About 10 c.c. clear juice with a few fine floculi. 5.15. Dog allowed to eat meat. Rate of secretion apparently not increased.

The animal stood whole procedure well and showed no ill effects afterwards. Died Nov. 13, 1907.

Dog 2. *Operation.* Simple gastric fistula. Wound healed *per primum.*

April 6, 1908. Dog fasted for past thirty-six hours. 11.15 A. M. Stomach catheterized; 0.9 c.c. of fluid removed. 11.45. Left external jugular vein exposed under light chloroform anesthesia and 5 c.c. of 0.7 per cent. NaCl injected intravenously. 11.50. Stomach catheterized; 3.3 c.c. of fluid removed. Dog allowed to run about. Not uncomfortable from the chloroform. 12 M. Stomach catheterized. No fluid removed. Showed and allowed to smell raw meat for 10 minutes. 12.10 P. M. Stomach catheterized; 4.3 c.c. of fluid removed. 12.15. Dog given full meal.

April 8, 1908. Dog fasted for past thirty-six hours. 2.45 P. M. Stomach catheterized; 3 c.c. of fluid removed (*specimen C*). 3.00. Stomach flushed with tap water. 3.10. Left external jugular vein exposed under light chloroform anesthesia and 8 c.c. submaxillary extract injected intravenously. (The glandular extract was prepared as follows: The two submaxillary glands were excised from a perfectly healthy dog under morphin-ether anesthesia, macerated with sterile sand in sterile 0.7 per cent. NaCl, and filtered through cloth.) 3.35. Catheterized; 9 c.c. of fluid removed (*specimen B*). 3.40. Showed and allowed to smell raw meat for ten minutes. 3.50. Catheterized; 9 c.c. of fluid removed (*specimen A*).

The three specimens of gastric fluid thus obtained were analyzed by one of us without knowledge of the foregoing procedure. *The results of this analysis were as follows:*

April 8, 1908. Analysis of gastric juice from dog with gastric fistula. 1 c.c. of the unfiltered juice was titrated with N/20 NaOH for the total acidity (phenolthalein) and free HCl (dimethylamidoazobenzol).

Specimen A. Total acidity = 1.95 c.c. N/20 NaOH
Free HCl = 1.30 c.c. "

Specimen B.	Total acidity = 0.90 c.c.	N/20 NaOH.
	Free HCl = 0.20 c.c.	"
Specimen C.	Total acidity = 1.26 c.c.	"
	Free HCl = 0.20 c.c.	"

Digestive power (Metz tubes); twelve hours at 39° C.

A (with addition of acid)	good digestion.
B (with " " ")	good "
C (with " " ")	good "
A (without " " ")	good "
B (without " " ")	no "
C (without " " ")	no "

C = material found in stomach after fasting.

Conclusion; B and C are identical.

April 10, 1908. Dog fasted for past thirty-six hours. 3.00 P. M. Catheterized; 8.6 c.c. of fluid removed containing much dark mucus (*specimen A*). 3.01. Deep chloroform anesthesia. Right external jugular vein exposed. 3.15. Catheterized; 3.4 c.c. of fluid removed (*specimen B*). 3.20; 5 c.c. of submaxillary extract injected intravenously. (The glandular extract was prepared as before.) 3.25. Catheterized; 8.8 c.c. of fluid removed (*specimen C*). 3.26. Showed and allowed to smell raw meat. 3.30. Catheterized; 5.3 c.c. of fluid removed (*specimen D*).

The results of chemical analysis, under the conditions as before, were as follows:

April 10, 1908. Four samples of gastric juice from dog with fistula. The titrations were done with N/20 NaOH, with phenolthalein for total acidity, with dimethylamidoazobenzol for free HCl.

Specimen A.	1 c.c., total acidity = 2.15 c.c.	N/20 NaOH.
	1 c.c., free HCl = 0.1 c.c.	"
Specimen B.	1 c.c., total acidity = 1.4 c.c.	"
	1 c.c., free HCl = 0.1 c.c.	"
Specimen C.	1 c.c., total acidity = 0.7 c.c.	"
	No free HCl	
Specimen D.	1 c.c., total acidity = 1.1 c.c.	"
	1 c.c., free HCl = 0.45 c.c.	"

Digestive power (Metz tubes); twelve hours at 39° C.

Without addition of acid.	With addition of HCl up to 2 per cent.
A — doubtful.	A — digestion.
B — "	B — very doubtful.
C — no digestion.	C — digestion.
D — digestion.	D — good digestion.

70 (326)

The relation of the weight of the stomach- and cecum-contents to the body weight in rabbits.

By **DON R. JOSEPH.**

[*From the Department of Physiology and Pharmacology of the Rockefeller Institute for Medical Research.*]

In much of our experimental work on mammals, we regulate our dosage in accordance with the weight of the animal. This is a source of error in the rabbit on account of the large size of the stomach and cecum in this animal. In order to find out how great this error is, I removed and weighed the contents of these two organs in one hundred rabbits which were used in the laboratory.

The details of these determinations will be given in the full report of this work. At present, I wish to say only that the average weight of the contents of these organs in the one hundred animals examined is equal to about ten per cent. of the total body weight of the living animal.

Grober[1] has recently made a similar series of observations, but he tied off, weighed and included, the stomach and cecum with their contents. According to my results, this is the source of another error which in some cases is almost as great as the inclusion of stomach- and cecum-contents in the body weight.

71 (327)

The inhibitory influence of magnesium upon some of the toxic effects of eserin.

By **DON R. JOSEPH.**

[*From the Department of Physiology and Pharmacology of the Rockefeller Institute for Medical Research.*]

In a paper on the relation of magnesium salts to gastro-intestinal peristalsis, Meltzer and Auer[2] stated that they were able to completely inhibit the general muscular tremor caused by eserin, by means of injections of magnesium sulfate.

Matthews and Jackson[3] stated later they did not obtain a like

[1] Grober: Untersuchungen zur Arbeitshypertrophie des Herzens. *Deutsches Arch. f. klin. Med.*, 1907, xci, p. 504.

[2] Meltzer and Auer: *Amer. Journ. Physiol.*, 1906, xvii, p. 317.

[3] Matthews and Jackson: *Amer. Journ. Physiol.*, 1907, xix, p. 12.

result. At Dr. Meltzer's suggestion, an attempt was then made to establish the facts in the case and to determine any other points of interest in the antagonism between these two drugs.

My results and methods may be briefly reported as follows:

First; a method of registering the general fibrillary muscular contractions was worked out, the best result being obtained by stitching a tambour to the skin over the gluteal muscles, with the leg in a flexed position.

Second; while writing the fibrillary tremor caused by eserin, $MgSO_4$[1] was slowly injected into the jugular vein. The result was an invariable stoppage of all tremor. This stoppage was brought about in from one half to one and a half minutes according to the rapidity of injection of magnesium. If the initial dose of $MgSO_4$ was sufficient, there was no recurrence of eserin tremor.

Third; an attempt was made to ascertain whether $MgSO_4$ could be used as an antidote for eserin poisoning. Eserin was injected into rabbits intramuscularly in quantities usually sufficient to kill. Afterwards, $MgSO_4$, was injected into the ear vein until the effect of eserin disappeared. In twenty two trials, the animals survived strongly toxic, and fatal, doses. The general tremor and convulsive movements stopped after the injection of from 2 to 4 c.c. of $MgSO_4$ solution. The magnesium effect was usually marked, however. If the depression of respiration was sufficient to cause danger of asphyxia, the injection of 1 c.c. of $CaCl_2$ solution into the ear vein was usually sufficient to counteract the action of $MgSO_4$ on the respiration.

Fourth; the myosis produced by instillation of eserin, is not removed or modified by any dose of magnesium.

72 (328)

Influence of iodides on autolysis.

By **L. B. STOOKEY.**

[*From the Physiological Laboratory, Medical School, University of Southern California.*]

Dogs were given 0.1 gram of KI per kilo daily over periods varying from one week to three months. Only in one case were

[1] In the use of magnesium sulfate, molecular solutions were employed in all cases.

typical symptoms of iodism prominent. The dogs were killed by bleeding from the carotids under ether anesthesia, the organs removed in the usual manner, hashed, divided into convenient portions, mixed with seven parts of saline and allowed to undergo autolysis at 37°C. in the presence of toluol. Kjeldahl determinations were made on the half saturated zinc sulphate filtrate and on the acidified saturated zinc sulphate filtrate.

In all cases the rate of autolysis was found to be considerably faster than that of organs taken from normal dogs. The *increased* rate of autolysis was noticeable particularly in the liver and kidneys, and especially during the first twenty four hours.

73 (329)

Relation of the thyroids to autolysis.

By **L. B. STOOKEY** and **VERA GARDNER.**

[*From the Physiological Laboratory, Medical School, University of Southern California.*]

Dogs were thyroidectomized. Typical symptoms of thyroid removal, appearing at variable intervals after the operation, were prominent in all the animals. From five to ten days after thyroidectomy, the dogs were killed by bleeding from the carotids under ether anesthesia, the organs removed, hashed, divided into convenient quantities, mixed with seven parts of saline and allowed to undergo autolysis at 37°C. in the presence of toluol. Kjeldahl determinations were made on the half saturated zinc sulphate filtrate and on the acidified saturated zinc sulphate filtrate.

In all cases the rate of autolysis of the organs from thyroidectomized dogs was slower than that of the organs from control animals. The *decreased* rate of autolysis was noticeable particularly in the liver and kidneys, and especially during the first twenty four hours.

74 (330)

On the physiology of the thyroids.

By **L. B. Stookey.**

[*From the Physiological Laboratory, Medical School, University of Southern California.*]

It seemed possible that many of the symptoms following thyroidectomy, namely, decreased metabolism, accumulation of mucoid in the subcutaneous connective tissues, colloidal changes in the kidneys and liver, hyaline degeneration in the arterial walls, decreased development of bones, delayed ossification of epiphysial cartilages and synchondroses, disturbances in temperature, and decline in muscular tone might result from decreased or incomplete protein oxidation. Conversely, this hypothesis would imply that the thyroids exert an influence over some intracellular oxidative processes.

To throw light on this hypothesis the power of tissues taken from normal animals to oxidize indol was compared with that of tissues taken from thyroidectomized dogs. The dogs were bled from the carotids, the organs removed under the usual precautions, hashed, divided into convenient quantities and exposed to seven times their weight of 0.005 per cent. indol solution. Toluol was added to prevent bacterial action. The mixtures were kept at body temperature, and the amount of unoxidized indol was determined at varying intervals. The indol was estimated colorimetrically by means of the glyoxylic acid reaction. Results obtained in this manner were checked by Kjeldahl nitrogen determinations.

In all cases it was found that thyroidectomy led to a *decreased* power on the part of the liver, kidneys, and spleen to oxidize indol, These findings might seem to indicate that the thyroids bear to intracellular nitrogenous oxidation a relation analogous to that existing between the pancreas and the utilization of glucose. namely: the thyroids through their internal secretion activate the oxidizing enzymes of the different body cells.

75 (331)

On the pharmacology of the iodides.

By **L. B. STOOKEY** and **VERA GARDNER.**

[*From the Physiological Laboratory, Medical School, University of Southern California.*]

In an earlier paper it was suggested by one of us that the internal secretion of the thyroids may influence metabolism in the manner of a kinase, namely, by activating some intracellular oxidases.

In view of a close parallelism existing between the iodine content of the thyroids and the physiological action of thyroid preparation it seemed possible that the administration of iodides might lead to an increased power to effect certain oxidative changes.

To test this hypothesis, the capacity of tissue taken from normal dogs to oxidize indol was compared with that of tissues taken from dogs treated with potassium iodide (0.1 gram per kilo daily) over variable periods. The dogs were bled from the carotids, the organs removed under the usual precautions, hashed, divided into convenient quantities and exposed to seven times their weight of 0.005 per cent. indol solution. Toluol was added to prevent bacterial action. The mixtures were kept at body temperature, and the amount of unoxidized indol was determined at varying intervals. The indol was estimated colorimetrically by means of the glyoxylic acid reaction. Results obtained in this manner were checked by Kjeldahl nitrogen determinations.

In all cases the administration of potassium iodide in the dog was found to lead to an *increased* power on the part of the liver, kidneys and spleen to oxidize indol.

While the influence of iodides upon intracellular oxidation might appear to be exerted through the thyroids, it is possible that a direct action without the intervention of the thyroids may take place. In this connection it is scarcely necessary to remark that the striking similarity between the action of iodides and that of thyreoglobulin was recognized long ago. Many symptoms of iodism are very similar to those of thyroidism, namely, tachycardia,

palpitation of the heart, muscular tremors, nervousness, sleepiness, and increased metabolism. A further study of the problem whether or not the increased oxidizing power of the different body cells following iodide administration is dependent upon the thyroids, is being made.

76 (332)

Glycogen formation from arabinose in chicks.

By **L. B. STOOKEY** and **A. HALDEN JONES.**

[*From the Physiological Laboratory, Medical School, University of Southern California.*]

It is well known that newly hatched chicks are practically free from glycogen. Fifteen chicks were fed arabinose over periods varying from several hours to two days, and their bodies examined for glycogen. In one case a trace of glycogen seemed to be present, but in all others negative results were obtained. However, these experiments are not looked upon as conclusive. Further studies are in progress.

77 (333)

Is oxalic acid a product of hepatic uricolysis in man?

By **L. B. STOOKEY** and **ETHEL L. LEONARD.**

[*From the Laboratories of Physiology and Bacteriology, Medical School, University of Southern California.*]

A liver obtained, shortly after death, from the accident ward of the City Hospital was hashed, divided into convenient portions and mixed with seven volumes of 0.1 per cent. solution of potassium urate. After varying periods, the mixtures were examined for oxalic acid.

In all cases the uric acid was found to be largely destroyed. In all cases perceptible traces of oxalic acid were shown to be present, yet the amounts isolated did not seem to be appreciably greater than those occurring in control experiments.

It does not seem, therefore, that oxalic acid is a product of hepatic uricolysis in man.

78 (334)

The life cycle of paramecium.

By LORANDE LOSS WOODRUFF.

[*From the Sheffield Biological Laboratory of Yale University.*]

On May 1, 1907, a culture of *Paramecium aurelia* (*caudatum*) was started with a "wild" individual from a laboratory aquarium, and since that time up to the present (May 20, 1908) it has been under daily observation. Hay infusion was employed as a culture medium during the first few months of the life of the culture, but later the culture medium has been constantly varied by employing water from different sources and to this water has been added any material which might be encountered in the natural habitat of *Paramecium*. In every case precautions have been taken to sterilize the infusion before using it in order to obviate the possibility of contaminating the culture by introducing a live cyst of a "wild" *Paramecium*. Conjugation has not been possible in the direct line of the culture because the individuals of the culture have been isolated almost daily.

Under these conditions the culture has so far attained 490 generations. During the greater part of the year the average rate of division of the culture has been between one and two divisions per day, and not during a single ten-day period has it averaged as low as one division in two days. Thus no period of marked physiological depression has been indicated by the division rate, and no special stimuli have been found necessary to save the culture from extinction.

The results obtained up to the present time show that the life cycle of *Paramecium*, when subjected to a varied environment, may be much longer in duration of time and may comprise many more generations than when subjected to a constant culture medium of hay infusion, as has been the case in previous investigations in this field.

79 (335)

An examination of Bardach's new protein test.

By **EMILY C. SEAMAN** and **WILLIAM J. GIES.**

[*From the Laboratory of Biological Chemistry of Columbia University, at the College of Physicians and Surgeons.*]

In a recent paper, Bardach[1] drew attention to the fact that the formation of typical iodoform crystals from iodopotassium iodide and acetone in an alkaline solution may be prevented by albumin. In the presence of a sufficient proportion of albumin in such solutions, the production of characteristic hexagonal platelets or stellar masses of iodoform does not occur, but, instead, fine *yellow needles*, apparently of some other substance, gradually appear. Bardach found that the power of bringing about this interesting crystalline reaction is also exhibited in a general way by acidalbumin, protoalbumoses, peptones, phytovitellin, casein, yeast nuclein, hemoglobin, tendomucoid, gelatin, and by the following protein-containing materials: pancreatin, sperm, blood, sputum, normal urine and albuminous urine. He did not name any proteins that fail to give the reaction.

The best conditions and procedure for the test are stated by Bardach to be as follows: To 5 c.c. of the moderately concentrated albuminous liquid,[2] add at first 2 or 3 drops of a dilute aqueous acetone solution (0.5 per cent.), then sufficient Lugol reagent[3] to supply a moderate excess of iodine,[4] and lastly *considerable* ammonium hydroxid (usually about 3 c.c. of concentrated solution).

If iodine is employed in *moderate* excess, the ammonium hydroxid usually produces at once a black precipitate of iodonitrocompounds, upon which the yellow needles are gradually deposited. If just the right amount of iodine is present, the liquid soon becomes yellowish and the black precipitate formed at once by the

[1] Bardach: *Zeitschrift für physiologische Chemie*, 1908, liv, p. 355.

[2] The proportion of protein should not exceed 5 per cent.

[3] Iodine (4 grams) and potassium iodide (6 grams) in water (100 c.c.).

[4] Sufficient to impart a permanent reddish brown color to the shaken solution. The required amount of Lugol reagent varies from 1 drop to several c.c. according to the proportion of protein, sugar or other iodine-reacting materials present.

ammonium hydroxid gradually undergoes complete or nearly complete transformation into the yellow needles. Under both conditions the yellow needles form promptly, usually within an hour. In the presence of too large an excess of iodine the reaction may be prevented or hidden in the heavy black precipitate of iodonitro-compounds. Too little iodine or too much protein also prevents the reaction.[1]

We have confirmed practically all the statements made by Bardach regarding the conditions affecting the production, as well as the chemical and physical characters, of the yellow needles. Bardach is convinced that, in spite of their iodoform-like odor and color, the yellow needles do not consist of iodoform but of a hitherto undescribed iodo-compound, which he proposes to prepare in abundance for detailed investigation. In harmony with Bardach's statements we have found the crystals practically insoluble in cold dilute mineral acids and alkalies, but somewhat soluble in cold water. In alcohol, ether and chloroform the crystals apparently undergo gradual modification into iodoform and probably other products.

We have made no special effort to advance in this study in any direction beyond the limits reached by Bardach himself, except to ascertain whether other proteins than those named by Bardach respond to the test. Applying the test as detailed above, we have obtained strikingly positive results with the following *additional* proteins and protein-containing materials :

* myosin
* edestin
* alkali albuminate (from myosin)
* acidalbumin (from muscle stroma substance)
* heteromucoses
deuteromucoses
elastoses
tendomucoid digestive products (peptic, soluble, mixed, including peptone)
* ligament mucoid
tendon mucoid (non-precipitable by acid)
bone mucoid (non-precipitable by acid)
* nucleoprotein (from ligament)
gelatins (from ligament and bones)
† osseoalbumoid
† chondroalbumoid
† ossein
† collagen-elastin (mixed ; from bones)

[1] When considerable protein and iodine are present, a *grayish green* precipitate is apt to form at once on adding ammonium hydroxid. If the proportions are favorable and the mixture is stirred continuously for a few minutes, the grayish green precipitate undergoes a gradual and very beautiful transformation into the glistening yellow needles.

* tendomucoid digestive residues (peptic)
ligament mucoid digestive products (peptic, soluble, mixed, including peptone)
* osseomucoids
* chondromucoids
† elastins (from ligament and tendon)
proteose-peptone (Witte's)
egg yolk (aqueous emulsion)
synovia
serum (dog blood)
lime water extract of tendon

* In dilute ammoniacal solution.

† Dilute solution, chiefly of *hydration products*, obtained by heating in dilute ammonium or potassium hydroxid.

It was found, on allowing the crystalline mixtures to stand for several days, that each precipitate diminished in amount very perceptibly and, also, on longer standing, that the crystals lost their characteristic canary-yellow color; they became opaque and when viewed through a microscope their outlines were less distinct than immediately after their formation.

The needle-like crystals obtained with the above named protein materials differed somewhat in character. The needles derived from some proteins were much finer and sharper than others. In some cases the needles were hair-like in appearance. As a rule the crystals were the same in each test with a given protein. Many of the crystals arranged themselves in rosettes or in bundle-like clusters; a few had a knobbed structure which gave them the appearance of nails. Some of the needles were branched like twigs of evergreen. It was a question whether the type of crystal was characteristic of the particular protein, or whether the differences in crystal-form were due to variations in the rapidity or other physical conditions of formation. It is probable, of course, that variations in the proportions of the reagents employed may have introduced chemical factors affecting the nature of the crystalline products. Iodoform crystals were mixed with the needle-like crystals whenever the proportion of protein was relatively low.

Whether Bardach's reaction is exhibited also by any non-protein substances has not been determined. It is probable, from the results of our own work, that without exception every soluble protein will give it. The test is quite delicate and promises to be useful in many ways, but its comparative value, and the disturbing influences in its application,[1] as a protein test await determination.

[1] Bardach has already noted a disturbing influence of earthy phosphate. Bardach: *loc. cit.*, p. 357.

80 (336)

A study of metabolic effects of experimental polycythemia in dogs.

By **WILLIAM WEINBERGER** (by invitation).

[*From the Laboratory of Biological Chemistry of Columbia University, at the College of Physicians and Surgeons.*][1]

Although the clinical and morphological signs of *plethora vera* are known to a large extent, the metabolic effects produced by increasing the amount of blood in the body have been but sparingly investigated. The present research deals with the latter question. The augmentation of the supply of blood was effected by intravenous transfusion of defibrinated blood taken from the same species. In one experiment direct transfusion was performed after connecting a femoral artery of one dog with a small branch of a saphenous vein in another. After each transfusion, as will be shown later, there developed a condition of the blood that closely resembled polycythemia, as observed in man. These experiments also bear upon certain phases of the general question of parenteral nutrition.

Seven experiments were conducted on as many apparently healthy dogs that were kept under observation for some time before the experiments were started. The majority of the experiments lasted from 5 to 6 weeks. Some dogs were under observation for even a longer time. Each experiment was divided into preliminary periods of normal nutritional conditions, and subsequent periods during which the metabolic influences of blood transfusion were studied. Two animals were put through a period of fasting that lasted 16 days in one case and 25 days in the other, during each of which two blood transfusions were performed to ascertain their possible nutritive value and metabolic effects. In some cases

[1] This series of experiments is the sixth of a group of reseaches, inaugurated in 1902 by Dr. Gies and now in progress in this laboratory, on the biochemical effects of change of volume of the circulating blood. See Gies: *American Medicine*, 1904, viii, p. 155; Posner and Gies: *Proceedings of the American Physiological Society: American Journal of Physiology*, 1904, x, p. xxxi; Hawk and Gies: *ibid.*, 1904, xi, p. 171; Meyer and Gies: *Proceedings of the Society for Experimental Biology and Medicine*, 1904, i, p. 44; Weingarten and Crohn: *American Journal of Physiology*, 1908, xxii, p. 207.

the effect of the ether narcosis was determined as was also the influence on metabolism of the operation itself without the transfusion. The body weight, the amount, specific gravity, and reaction of the urine, and the weight of the dried feces were noted in the daily record. In all experiments the total amounts of nitrogen that were ingested and the corresponding totals that were excreted in the urine were determined; in some, the total sulfur and phosphorus intakes and outputs were ascertained in addition. The urine was examined for coagulable protein, urobilin, blood and casts. In a special experiment a microscopic examination of the blood was made to determine the variations in the number of erythrocytes. The specific gravity was also ascertained by means of Schmaltz's pyknometer and the presence or absence of hemoglobinemia determined.

In all cases autopsies were performed and in one animal, that showed the metabolic changes especially well during life, a microscopic examination of the liver and kidneys was made. In order to obtain emphatic results in the determination of the metabolic effect of blood transfusion, it was necessary to transfer rather large quantities. The amount of transported blood ranged between 3.5 per cent. and 7.5 per cent. of the body weight at each transfusion. It was also found expedient to perform a number of transfusions at comparatively short intervals (from 6 to 8 days) in order to develop cumulative effects, if possible. For the purpose of checking the metabolic influence of distention of the vascular system and its filling up with liquid material, infusions of 0.9 per cent. NaCl solution were performed in the same manner and under the same conditions as the blood transfusions. In order fully to understand the metabolic changes produced by blood transfusion, it is necessary to ascertain the effects of serum and corpuscles separately, a task which has been made the object of a series of experiments now in progress.

The results obtained so far may be summarized briefly as follows:

Transfused blood does not remain in its entirety in the vascular system, but, as indicated above, its several constituents are disposed of unequally. The first to be removed is the water, which fact accounts for the greatly increased volumes of urine

excreted during the first few days after transfusion. However, the diuresis following transfusion of blood is very much less than that produced by infusion of 0.9 per cent. NaCl solution. In the case of the latter, the total volume infused appears in the urine of the succeeding twenty four hours. Nevertheless, the diuretic effect of blood transfusion is quite marked.

The excretion of nitrogen in the urine was decidedly increased by the transfusion of blood, the rise becoming more and more pronounced with each successive transfusion. To cite one instance: The average daily urinary nitrogen rose from 6.28 grams in the preliminary period of nitrogen equilibrium to 10.23 grams after the 4th blood transfusion. To what extent the different constituents of the blood contribute to this result; whether the increase of nitrogen is derived from the blood (the serum or corpuscles), or from the body proteins destroyed by any toxic action of the transfused material, or whether the increase is caused by both (the most probable thing) will be investigated later.

"Blood counts" show that the number of erythrocytes per unit of volume rises with each transfusion, which fact is due to the elimination of water, the red cells being retained in the vascular system a much longer time, in this manner causing a distinct polycythemia. By a series of transfusions it was possible to increase the number of erythrocytes from 7,272,000 to 13,512,000 in one cmm. In this connection it is interesting to note that, after rising, the "blood count" showed a decline which set in after a few days. Then another transfusion was performed, and the number of red cells increased still further. In this manner a continued and more and more pronounced polycythemia was produced. The specific gravity of the blood rose also with each transfusion. It is noteworthy, also, that after a few transfusions the specific gravity was relatively higher as compared with the number of red cells, showing a greater concentration of the blood serum proper. The specific gravity rose after a few transfusions from 1.066 to 1.103.

No decided increase in the nitrogen output has been observed after saline infusion. The daily urinary output of nitrogen from the control dog increased from 5.29 grams in the preliminary period of nitrogenous equilibrium to 5.91 grams after the *ninth* saline infusion.

The excretion of total sulfur in the urine has been found to run approximately parallel with that of nitrogen. The fluctuations in the amounts of total phosphorus in the urine do not allow of any definite deductions at the present time.

The defibrinated blood, after being filtered by suction, was in all cases moderately hemolytic, a change produced by the act of defibrination. Nevertheless, in the great majority of cases the urine passed after transfusion did not contain any hemoglobin, the organism of the dog apparently disposing of the same, completely storing its derivatives in the different structures of the body, especially the liver, which on post mortem examination proved to be of a dark brown, almost black color and contained large amounts of hemosiderin in the ferric state, as shown by microchemical examination of sections. In some instances very concentrated bile was found in the gall bladder. Accordingly there was a decided increase in the coloring matter of the feces and especially in the urine, conditions which, after a series of blood transfusions, were very greatly emphasized, the urine containing a large amount of urobilin.

All experiments ended with the death of the animal, the 4th or 5th transfusion of blood invariably proving fatal. It was possible to predict close approach to the danger line from the appearance in the urine of coagulable protein. Coagulable protein never was found in the urine until after a third or fourth transfusion had been made; its appearance preceded a rapid decline of the animal, the urine examination showing the presence of granulated casts and epithelial cells. The succeeding transfusion usually sufficed to produce hemolysis accompanied by hemoglobinuria, from which condition the animal never recovered. On microscopic examination, a section from such a dog's kidney showed, besides enormous hemorrhagic areas, parenchymatous degeneration of the epithelial cells of the tubuli contorti. In these respects experimental polycythemia resembles the polycythemia occurring in human beings, the latter disease terminating fatally in almost all cases, the urine containing coagulable protein and granulated casts.

With reference to the nutritive value of blood transfusion hardly anything can be said in favor of it, the body weight which is naturally higher immediately after transfusion gradually sinking to or even below the initial level. Even in fasting animals the transfusion

of blood did not appreciably retard the daily average loss in body-weight, but actually quickened it as a rule.

The work is nearing completion and its results will shortly be published in detail.

81 (337)

On the metabolic influence of magnesium sulfate in dogs, with special reference to the partition of the nitrogenous constituents of the urine.

By **MATTHEW STEEL** (by invitation).

[*From the Laboratory of Biological Chemistry of Columbia University, at the College of Physicians and Surgeons.*]

In these experiments, in which relatively large doses of magnesium sulfate were given to dogs, abscesses and sloughing followed subcutaneous injections, but were not caused by intramuscular or intravenous injections nor by administrations *per os*. Very large doses of magnesium sulfate could repeatedly be injected intravenously without causing death, when care was taken to conduct the process slowly.

Daily fluctuations in the weight of the animals, as well as in the volume and specific gravity of their urines, resulted chiefly from diuretic or diarrheal influences and the consequent compensatory tendencies.

Administration *per os* caused diarrhea. Bone ash in the food appeared to exert only a mechanical diminution of such diarrheal tendencies. Injections under the skin or into a muscle or into the circulation failed to elicit any evidence of diarrhea, except in one doubtful case after subcutaneous application. On the contrary, such injections appeared to make the feces drier and harder than ordinarily, and the urine volumes greater.

If there was any effect on the quantitative elimination of solid matter in the feces, it was not more than a slight increase. The same may be said of the content of nitrogen in the feces, and also of the fecal discharge of magnesium (after intravenous injection of magnesium sulfate).

In a general way elimination of nitrogen in the urine was increased after the normal periods, but the increase was not suf-

ficient to warrant the conclusion that it was a direct effect of the dosage. The observed absolute increase of urinary nitrogen was registered chiefly in the form of urea, although the relative excretion of the latter was below normal in one of the two main experiments.

The most striking and consistent effect on the partition of the urinary nitrogen was the continued absolute as well as relative increase of ammonia elimination throughout the whole of the dosage part of each metabolism experiment, in spite of the fact that the Folin method does not permit of complete recovery of ammonia from crystallized ammonio-magnesium phosphate. (See the next abstract.) The increased elimination of ammonia nitrogen may be attributed, in large part at least, to special formation and elimination of ammonio-magnesium phosphate under the prevalent conditions.

Considering the data pertaining to the partition of nitrogen from the standpoint of direct agreement, in the two main experiments, it is noticeable that the increased elimination of ammonia in all the postnormal periods is the most striking and perfect concordance. That this increase was largely due to the combination of ammonia with magnesium in the form of ammonio-magnesium phosphate appears to be certain. The increase indicated by our figures in one of the two main experiments was doubtless below the full increase that would have been exhibited by a perfect method of determining ammonia nitrogen when in the form of "triple phosphate."

The nitrogen of urea, creatinin and allantoin, taken collectively, appeared in increased amounts, both absolutely and relatively, in a majority of the postnormal periods.

It is especially noteworthy that recovery from dosage with magnesium sulfate, however profound the immediate effect of such treatment may have been, was always prompt and apparently complete so far as general observation and our data indicated. That magnesium sulfate exercises surprisingly little measurable effect on nitrogen metabolism under the conditions of these experiments has also been shown by the results.

The paper will shortly appear in the *Journal of Biological Chemistry*.

82 (338)

On the determination of ammonia, by the Folin method, in urines containing crystalline ammonio-magnesium phosphate.

By **MATTHEW STEEL** and **WILLIAM J. GIES.**

[*From the Laboratory of Biological Chemistry of Columbia University, at the College of Physicians and Surgeons.*]

Last fall during the progress of the metabolism research that is described in the abstract immediately preceding this, certain anomalous results were obtained in our quantitative determinations of the urinary ammonia. In the earlier periods of that research, urinary ammonia had been determined, by the Folin method, in the urines in duplicate for 38 days, with thoroughly concordant results. Shortly after the beginning of a metabolism period, however, during which magnesium sulfate was injected subcutaneously every twenty four hours, the titrations in duplicate (at the conclusion of the Folin process as applied to the daily urines), were strikingly discordant, the disagreements amounting to from 1 to 2 c.c. of $n/5$ KOH per 25 c.c. of urine.

Our inability to obtain satisfactory duplicate results for urinary ammonia content after the magnesium sulfate treatment, or to explain the analytic discrepancies by any probable fault of technique, led us to make two general suppositions regarding the cause of the analytic disagreements observed:

1. That magnesium was eliminated into the urines in question in relatively large quantities as ammonio-magnesium phosphate, which separated, in part at least, in typically crystalline masses.

2. That the *crystalline* ammonio-magnesium phosphate thus deposited was not thoroughly decomposed by sodium carbonate, as used in the Folin process, whereby ammonia, in variable amounts, remained in its solid form as triple phosphate in the urines under investigation.

General examination of the urines that gave the anomalous quantitative results for ammonia content showed at a glance that our first supposition was correct — triple phosphate had crystallized in abundance. In separating portions of the urines for analysis,

care had always been taken to isolate fractions of the thoroughly shaken and even mixed daily samples. Consequently, we had no reason to believe that any of the above mentioned anomalous results of the ammonia determinations were due to transferral of unequal amounts of the deposited ammonio-magnesium phosphate in the duplicate fractions of the urine taken. We therefore proceeded to test very carefully, and in many trials, the validity of the second supposition stated above.

First Series. — Is the amount of sodium carbonate (1–2 grams) that is usually taken with 25 c.c. of urine in the Folin process sufficient to completely liberate the ammonia from small quantities of crystalline ammonio-magnesium phosphate?

We endeavored to answer this question directly by the following special adaptation of the Folin process: Portions of pure, crystalline ammonio-magnesium phosphate, in different amounts between 50 and 500 milligrams inclusive, were quickly and very accurately weighed on a watch glass and transferred quantitatively to aerometer cylinders of the usual size, through a small dry funnel from which the tube had been removed. All fragments adherent to the watch glass and funnel were brushed into the cylinders. No losses of substance could have occurred in the process. In all the tests the crystalline matter was a comparatively coarse powder. About 25 to 50 c.c. of water were poured into the cylinders onto the powder, which quickly formed a loose sediment in the undisturbed water. A layer of kerosene was then poured over the liquid in each cylinder merely to duplicate the conditions of the Folin process although no special frothing could have occurred to require its use. Solid sodium carbonate in definite quantities ranging between 1 and 4 grams inclusive, was then added to the phosphate-water-kerosene mixture in the cylinder. The apparatus recommended by Folin was employed for aeration. More powerful pumps than those recommended by Folin were kept in operation for from five to fifteen hours, so that aeration was unusually effective. In all cases aeration was continued at least five hours. In the groups designated *B* and *C* (Table I) aeration was conducted during a second five-hour period, or ten hours in all. The aerometer cylinders were not opened between the two periods, but the acid of the first period of absorption was removed and a new

portion of acid substituted for ammonia absorption during the second aeration period. In the sixth determination (Group *B*) aeration was carried in the same manner through a third period of five hours, or fifteen hours in all. Approximately fifth-normal sulfuric acid was used for the absorption of the ammonia. Congo red was used as the indicator. Our results in the first series of tests are given in Table I.

TABLE I.

First Series. Groups A–C.

Pure, crystalline ammonio-magnesium phosphate,[1] 0.05–0.5 gram. Sodium carbonate, 1–4 grams. Total periods of aeration, 5–15 hours. Loss of ammonia; maximum, 48.1 per cent.; minimum, 31.1 per cent.

Group.	Determination. No.	Weight of NH_4MgPO_4. Gram.	Weight of Na_2CO_3. Grams.	Volume of Standard Acid Solution Required to Neutralize.					Ammonia Lost, Per Cent.
				After Aeration.				If All NH_3 Had Been Liberated,[2] c.c.	
				5 Hrs., c.c.	5 Hrs., (2) c.c.	5 Hrs., (3) c.c.	Total, c.c.		
A	1	0.05	1	0.6	—	—	0.6	1.05	42.9
	2	0.1	1	1.2	—	—	1.2	2.05	41.5
	3	0.2	1	2.4	—	—	2.4	4.10	41.5
	4	0.3	1	4.2	—	—	4.2	6.15	31.7
B	5	0.4	4	4.1	0.55	—	4.65	8.25	43.6
	6	0.5	4	4.6	1.20	1.30	7.10	10.30	31.1
C	7	0.5	1	4.3	1.05	—	5.35	10.30	48.1
	8	0.5	2.5	5.1	0.80	—	5.90	10.30	42.7
	9	0.5	4	5.6	0.65	—	6.25	10.30	39.3

The results in Table I show that 1 gram of sodium carbonate was unable, after five hours of very strong aeration, completely to eject the ammonia from 50 milligrams of the triple phosphate. The data for Groups *B* and *C* show that, after ten hours' aeration of 0.4 or 0.5 gram samples of triple phosphate with 1 to 4 grams of sodium carbonate, large proportions of ammonium were undisturbed in the crystalline material. Even after fifteen hours' aeration of a 0.5 gram sample of the phosphate with 4 grams of sodium

[1] NH_4MgPO_4, $6H_2O$. A pure crystalline powder obtained from Eimer and Amend. The theoretical content of nitrogen is 5.707 per cent. The average result of seven closely concordant determinations of nitrogen content by the Kjeldahl method was 5.727 per cent. (5.697, 5.766, 5.780, 5.669, 5.725, 5.725, 5.725).

[2] Seven determinations of the volume of our standard acid solution that was required to neutralize the ammonia liberated in the Kjeldahl process from 0.5 gram samples of the triple phosphate used in this series gave the following results (c.c.): 10.25, 10.40, 10.40, 10.20, 10.30, 10.30, 10.30, or an average of 10.30 c.c.

carbonate (6), practically one third of the ammonium remained undisplaced.

These observations gave strong support to our second conclusion regarding the cause of the anomalous ammonia results that prompted this study.

Eighth Series.—This investigation was concluded with a determination of the effects of relatively very great excesses of sodium carbonate in the aeration process. The results of this final test, which are given in Table II, merely confirmed the conclusion already drawn that Folin's splendid method fails, in the case of triple phosphate, to give perfectly accurate results for ammonia content.

TABLE II.

Eighth Series. Group R.

Pure, crystalline ammonio-magnesium phosphate (Eimer & Amend product), 0.5 gram. Sodium carbonate, 2–16 grams. Periods of aeration (2), 5 hours. Loss of ammonia: maximum, 35.19 per cent; minimum, 12.04 per cent.

Group.	Determination. No.	Weight of NH_4MgPO_4 Gram.	Weight of Na_2CO_3 Grams.	Volume of Standard Acid Solution Required to Neutralize.				Ammonia Lost, Per Cent.
				After Aeration.			If All NH_3 had been Liberated, c.c.	
				5 Hrs., c.c.	5 Hrs., (2) c.c.	Total, c.c.		
R	85	0.05	2	0.65	0.05	0.70	1.08	35.19
	86	0.05	4	0.60	0.15	0.75	1.08	30.56
	87	0.05	8	0.70	0.10	0.80	1.08	25.93
	88	0.05	16	0.80	0.15	0.95	1.08	12.04

Our paper on this subject will soon be published in the *Journal of Biological Chemistry.*

RECAPITULATION OF THE NAMES OF THE AUTHORS AND OF THE TITLES OF THE COMMUNICATIONS.

VOLUME V.

Adler, Isaac [with **O. Hensel.**]

282. Studies in experimental arteriosclerosis.

Atkinson, James P. [with **Arthur I. Kendall.**]

267. A modification of Teichmann's method for obtaining hemin crystals, with a demonstration of specimens.

Auer, John

263. [With **S. J. Meltzer.**] Effects of calcium and magnesium salts upon the development of rigor mortis.

272. Gastric peristalsis after section of the vagi and splanchnic nerves.

274. [With **S. J. Meltzer.**] The antagonistic action of calcium upon the inhibitory effect of magnesium.

305. [With **S. J. Meltzer.**] The action of calcium upon the pupil and its relation to the effects of mydriatics.

Bailey, Harold C. [with **Robert A. Hatcher.**]

306. The destruction of strophanthin in the animal organism.

Banzhaf, Edwin J.

269. [With **Edna Steinhardt.**] The relative value of antitoxin and other curative substances in antidiphtheric serum.

291. [With **L. W. Famulener.**] A note on anaphylaxis.

Brooks, Harlow [with **B. C. Crowell.**]

276. Concerning the relation of the coagulation time of the blood to thrombosis in phlebitis.

Brown, Amos P. [with **Edward T. Reichert.**]

293. The crystallography of hemoglobins.

Brown, E. D. [with **Torald Sollmann.**]

266. A depressor reaction obtainable by traction on the carotid artery.

Burton-Opitz, Russell

279. [With **Daniel R. Lucas.**] On the circulation through

the kidneys. I. On vasomotor reactions. II. The renal blood flow in relation to the pressure in the ureter and bladder. III. The effect of solutions of adrenalin.

280. Some data regarding the portal circulation.

281. A clinical viscosimeter.

311. [With **Daniel R. Lucas.**] Regarding the innervation of the blood vessels of the kidney.

312. Regarding the innervation of the blood vessels of the intestine.

Carrel, Alexis

265. Reëstablishment of function in transplanted kidneys.

275. Remote result of the transplantation of a segment of popliteal artery from a man to a bitch.

Cohen, L. J. [with **William J. Gies.**]

314. A study of "protagon" prepared by the Wilson-Cramer method.

Cooke, Elizabeth [with **Leo Loeb.**]

270. The effect of light on cells in fluorescent solution after addition of potassium cyanide.

317. Hemolytic action of the venom of *Heloderma suspectum.*

Crampton, C. Ward

271. Physiological age.

Crampton, Henry E.

287. Physiological problems of the geographical distribution of *Partula* in Polynesia, with demonstration of specimens.

Crowell, B. C. [with **Harlow Brooks.**]

276. Concerning the relation of the coagulation time of the blood to thrombosis in phlebitis.

Davenport, Charles B.

315. Heredity of some human physical characteristics.

Dunham, Edward K.

288. Note on the isolation of carnaubic acid from beef kidneys.

Elsberg, Charles A.

259. Pneumothorax and posture.

Emerson, Haven

257. Cardiac insufficiency due to high arterial pressure.

Famulener L. W. [with **Edwin J. Banzhaf.**]

291. A note on anaphylaxis.

Field, Cyrus W.

290. Further observations on the precipitation of inorganic colloids by sera.

Flexner, Simon

264. [With **James W. Jobling.**] Restraint and promotion of tumor growth.

284. [With **James W. Jobling.**] Metaplasia and metastasis of a rat tumor.

308. Prevention of syphilis in *Macacus Rhesus* by atoxyl.

309. [With **James W. Jobling.**] Further notes on a rat tumor.

Foster, Nellis B. [with **Adrian V. S. Lambert.**]

319. Variation in hydrochloric acid secretion during the digestive period.

320. The effects of some organic acids on the secretion of gastric juice.

321. The effect of mechanical obstruction of the pyloric outlet on gastric secretion.

Gardner, Vera [with **L. B. Stookey.**]

329. Relation of the thyroids to autolysis.

331. On the pharmacology of the iodides.

Gay, Frederick P.

262. The rôle of tonicity in human isohemagglutination.

289. The change of corpuscle resistance in the blood of immunized animals, coincident with the formation of anti-bodies.

302. [With **E. E. Southard.**] The relative specificity of anaphylaxis.

Gies, William J.

314. [With **L. J. Cohen.**] A study of "protagon" prepared by the Wilson-Cramer method.

335. [With **Emily C. Seaman.**] An examination of Bardach's new protein test.

338. [With **Matthew Steel.**] On the determination of ammonia, by the Folin method, in urines containing crystalline ammonio-magnesium phosphate.

Halsted, W. S.

297. The transplantation of parathyroid glands in dogs.

Hatcher, Robert A. [with **Harold C. Bailey.**]

306. The destruction of strophanthin in the animal organism.

Hawk, Philip B.

268. The influence of ether anesthesia on the excretion of nitrogen by dogs.

Hensel, O. [with **Isaac Adler.**]

282. Studies in experimental arteriosclerosis.

Hooker, D. R. [with **A. S. Loevenhart.**]

325. Note upon the supposed presence of a gastric hormon in the salivary glands.

Imchanitzky, Marie

298. The nervous coördination of the auricles and ventricle of the heart of the lizard.

[Communicated by **S. J. Meltzer.**]

Jacobs, W. A. [with **John A. Mandel** and **P. A. Levene.**]

310. On nucleic acids.

Janeway, Theodore C. [with **Herman O. Mosenthal.**]

323. A study of nitrogen metabolism in a case presenting short paroxysms of fever of unknown origin.

Jobling, James W. [with **Simon Flexner.**]

264. Restraint and promotion of tumor growth.

284. Metaplasia and metastasis of a rat tumor.

309. Further notes on a rat tumor.

Jones, A. Halden [with **L. B. Stookey.**]

332. Glycogen formation from arabinose in chicks.

Joseph, Don R.

273. [With **S. J. Meltzer.**] The effect of stimulation of the vagus nerves upon the development of rigor mortis of the mammalian heart.

304. [With **S. J. Meltzer.**] The relative toxicity of the chlorides of magnesium, calcium, potassium and sodium.

326. The relation of the weight of the stomach- and cecum-contents to the body weight in rabbits.

327. The inhibitory influence of magnesium upon some of the toxic effects of eserin.

Kendall, Arthur I. [with **James P. Atkinson.**]

267. A modification of Teichmann's method for obtaining hemin crystals, with a demonstration of specimens.

Lambert, Adrian V. S. [with **Nellis B. Foster.**]

319. Variation in hydrochloric acid secretion during the digestive period.

320. The effects of some organic acids on the secretion of gastric juice.

321. The effect of mechanical obstruction of the pyloric outlet on gastric secretion.

Larkin, John H. [with **Isaac Levin.**]

322. Transplantation of devitalized arterial segments.

Leonard, Ethel L. [with **L. B. Stookey.**]

333. Is oxalic acid a product of hepatic uricolysis in man?

Levene, P. A. [with **John A. Mandel** and **W. A. Jacobs.**]

310. On nucleic acids.

Levin, Isaac

277. The reactive power of the white rat to tissue implantation. Second communication.

322. [With **John H. Larkin.**] Transplantation of devitalized arterial segments.

Lewis, Paul A.

260. The hypersensitiveness of the guinea-pig to horse serum.

313. Note on anaphylaxis in horse serum.

Loeb, Leo

270. [With **Elizabeth Cooke.**] The effect of light on cells in fluorescent solution after addition of potassium cyanide.

316. The experimental production of the material part of the placenta in the rabbit.

317. [With **Elizabeth Cooke.**] Hemolytic action of the venom of *Heloderma suspectum.*

Loevenhart, A. S. [with **D. R. Hooker.**]

325. Note upon the supposed presence of a gastric hormon in the salivary glands.

Lucas, Daniel R. [with **R. Burton-Opitz.**]

279. On the circulation through the kidneys. I. On vasomotor reactions. II. The renal blood flow in relation to the pressure in the ureter and bladder. III. The effect of solutions of adrenalin.

311. Regarding the innervation of the blood vessels of the kidney.

Lusk, Graham

295. Influence of cold and exercise in phlorhizin glycosuria.

MacCallum, W. G. [with **Carl Voegtlin.**]

303. On the relation of calcium metabolism to tetany and the cure of tetany by administration of calcium.

MacLeod, J. J. R.

307. On the nature of the so called glycogenolytic fibers in the greater splanchnic nerves.

Mandel, John A. [with **W. A. Jacobs** and **P. A. Levene.**]

310. On nucleic acids.

McCoy, George W. [with **M. J. Rosenau.**]

294. The germicidal property of milk.

Meltzer, S. J.

263. [With **John Auer.**] Effects of calcium and magnesium salts upon the development of rigor mortis.

273. [With **Don R. Joseph.**] The effect of stimulation of the vagus nerves upon the development of rigor mortis of the mammalian heart.

274. [With **John Auer.**] The antagonistic action of calcium upon the inhibitory effect of magnesium.

298. The nervous coördination of the auricles and ventricle of the heart of the lizard. [Communicated for **Marie Imchanitzky.**]

304. [With **Don R. Joseph.**] The relative toxicity of the chlorides of magnesium, calcium, potassium and sodium.

305. [With **John Auer.**] The action of calcium upon the pupil and its relation to the effects of mydriatics.

Mendel, Lafayette B.

299. The influence of diet on the chemical composition of the body.

300. [With **Tadasu Saiki.**] The chemical composition of nonstriated mammalian muscle.

Morgan, T. H.

286. The production of two kinds of spermatozoa in phylloxerans — functional "female producing" and rudimentary spermatozoa.

Mosenthal, Herman O. [with **Theodore C. Janeway.**]

323. A study of nitrogen metabolism in a case presenting short paroxysms of fever of unknown origin.

Murlin, J. R.

296. The influence of carbohydrate on the protein metabolism of a fasting pregnant dog.

Osborne, Thomas B.

318. The biological relations of seed proteins.

Reichert, Edward T. [with **Amos P. Brown.**]

293. The crystallography of hemoglobins.

Richards, A. N. [with **George B. Wallace.**]

258. Effect of potassium cyanide upon metabolism.

Robertson, T. Brailsford

283. On the influence of various substances, applied directly to the medulla oblongata, upon the respiratory rhythm in frogs.

Rosenau, M. J. [with **George W. McCoy.**]

294. The germicidal property of milk.

Saiki, Tadasu [with **Lafayette B. Mendel.**]

300. The chemical composition of nonstriated mammalian muscle.

Seaman, Emily C. [with **William J. Gies.**]

335. An examination of Bardach's new protein test.

Sollmann, Torald [with **E. D. Brown.**]

266. A depressor reaction obtainable by traction on the carotid artery.

Southard, E. E. [with **F. P. Gay.**]

302. The relative specificity of anaphylaxis.

Steinhardt, Edna [with **Edwin J. Banzhaf.**]

269. The relative value of antitoxin and other curative substances in antidiphtheric serum.

Steel, Matthew

337. [By invitation.] On the metabolic influence of magnesium sulfate in dogs, with special reference to the partition of the nitrogenous constituents of the urine.

338. [With **William J. Gies.**] On the determination of ammonia, by the Folin method, in urines containing crystalline ammonio-magnesium phosphate.

Stookey, L. B.

328. Influence of iodides on autolysis.

329. [With **Vera Gardner.**] Relation of the thyroids to autolysis.

330. On the physiology of the thyroids.

331. [With **Vera Gardner.**] On the pharmacology of the iodides.

332. [With **A. Halden Jones.**] Glycogen formation from arabinose in chicks.

333. [With **Ethel L. Leonard.**] Is oxalic acid a product of hepatic uricolysis in man?

Tyzzer, E. E.

261. A sporozoan found in the peptic glands of the common mouse.

Voegtlin, Carl [with **W. G. MacCallum.**]

303. On the relation of calcium metabolism to tetany and the cure of tetany by administration of calcium.

Wallace, George B. [with **A. N. Richards.**]

258. Effect of potassium cyanide upon metabolism.

Ward, Wilbur

324. Histological changes in transplanted blood vessels. [Communicated by **Francis Carter Wood.**]

Weil, Richard

278. The hemolytic reactions of the blood in dogs with transplantable lymphosarcoma.

Weinberger, William [by invitation.]

336. A study of metabolic effects of experimental polycythemia in dogs.

Wilson, Edmund B.

285. An exhibition of photographs of chromosomes, with explanatory comment.

Wood, Francis Carter

324. Histological changes in transplanted blood vessels. [Communicated for **Wilbur Ward.**]

Woodruff, Lorande Loss

301. Increased susceptibility of protozoa to poison due to treatment with alcohol.

334. The life cycle of *Paramecium.*

Yerkes, Robert M.

292. The relation of plasticity to sex and age in the dancing mouse.

CLASSIFIED LIST OF CONTRIBUTING LABORATORIES.

(Communications in Volume V.)

Adler's private laboratory — 282.[1]

Carnegie Institution's Station for Experimental Evolution — 315.

Columbia University : *Biological Chemistry* — 314, 319, 320, 321, 323, 335, 336, 337, 338 ; *Clinical Pathology* — 324 ; *Pathology* — 277, 322 ; *Pharmacology* — 258 ; *Physiology* — 257, 279, 280, 281, 311, 312 ; *Zoölogy* — 285, 286, 287.

Connecticut Agricultural Experiment Station — 318.

Cornell University Medical College : *Huntington Fund for Cancer Research* (of the General Memorial Hospital, Loomis Laboratory) — 278 ; *Pharmacology* — 306.

Danvers Insane Hospital — 262.

Harvard University : *Caroline Brewer Croft Cancer Commission* — 261 ; *Pathology* — 289, 302 ; *Psychology* — 292.

Johns Hopkins University : *Hunterian Laboratory* — 297, 303 ; *Physiology* — 325.

Massachusetts State Board of Health, Antitoxin Laboratory — 260, 313.

New York Board of Education, Department of Physical Training — 271.

New York Department of Health : *Chemical Laboratory* — 267 ; *Research Laboratory* — 269, 290, 291.

New York University and Bellevue Hospital Medical College : *Pathology* — 276, 288 ; *Physiology* — 295, 296.

Rockefeller Institute for Medical Research : 264, 265, 275, 284, 288, 308, 309, 310 ; *Physiology and Pharmacology* — 259, 263, 272, 273, 274, 304, 305, 326, 327.

U. S. Public Health and Marine Hospital Service, Hygienic Laboratory — 294.

University of Berne, Physiological Institute — 298.

University of California, Rudolph Spreckles Physiological Laboratory — 283.

University of Pennsylvania : *Experimental Pathology* — 270, 316, 317 ; *Physiological Chemistry* — 268 ; *Physiology* — 293.

University of Southern California, Physiological Laboratory — 328, 329, 330, 331, 332, 333.

Western Reserve University : *Pharmacology* — 266 ; *Physiology* — 307.

Yale University : *Biology* — 301, 334 ; *Physiological Chemistry* — 299, 300.

[1] The numerals correspond with those in parenthesis above the titles of the abstracts (pages 1–134).

EXECUTIVE PROCEEDINGS.

QUOTATIONS FROM THE MINUTES.

Twenty fifth meeting.

College of Physicians and Surgeons, Columbia University. October 16, 1907. President Flexner in the chair.

Members present: Adler, Auer, Brooks, Burton-Opitz, Carrel, Crampton (C. W.), Emerson, Ewing, Field, Flexner, Foster, Gay, Gies, Hatcher, Jobling, Kast, Lee, Levene, Levin, Lewis, Lusk, Mandel (A. R.), Mandel (J. A.), Meltzer, Morgan, Murlin, Noguchi, Oertel, Park, Richards, Terry, Teague, Tyzzer, Wallace, Weil, Wolf, Wood.

Members elected: Edward T. Reichert, M. J. Rosenau, Richard P. Strong.

Twenty sixth meeting.

Rockefeller Institute for Medical Research. December 18, 1907. President Flexner in the chair.

Members present: Adler, Auer, Berg, Burton-Opitz, Brooks, Carrel, Crampton (C. W.), Ewing, Field, Flexner, Gies, Lee, Levene, Levin, Loeb (L.), Meltzer, Murlin, Norris, Salant, Terry, Weil.

Members elected: Edwin J. Banzhaf, William N. Berg, H. D. Dakin, Charles A. Elsberg, Shinkishi Hatai, Charles E. Simon, S. Burt Wolbach.

Twenty seventh meeting.

[Fifth annual business meeting.]

Schermerhorn Hall, Columbia University. February 19, 1908. Vice-President Morgan in the chair.

Members present: Auer, Banzhaf, Crampton (H. E.), Dunham, Famulener, Gager, Gay, Gies, Joseph, Kast, Lusk, Meltzer, Morgan, Murlin, Sherman, Teague, Wilson.

Members elected: L. W. Famulener, C. Stuart Gager, W. S. Halsted, Walter A. Jacobs, Theodore C. Janeway, Don R. Joseph, Frank R. Lillie, Alwin M. Pappenheimer, Donald D. Van Slyke.

Officers elected: President, Lee; Vice-president, Morgan; Treasurer, Lusk; Secretary, Gies.

Treasurer's report: The main items of the retiring Treasurer's report for the year 1907–'08 were the following:

Balance in the Treasury, February 20, 1907	$ 28.18	
Receipts	426.94	$455.12
Expenditures		408.10
Balance of cash on hand		47.02
Bills payable	116.39	
Bills receivable	102.08	

Twenty eighth meeting.

New York University and Bellevue Hospital Medical College. April 15, 1908. President Lee in the chair.

Members present: Auer, Carrel, Elsberg, Famulener, Flexner, Foster, Gay, Gies, Halsted, Hatcher, Jacobs, Jobling, Joseph, Lee, Levene, Lusk, MacCallum (W. G.), Mandel (A. R.), Mandel (J. A.), Meltzer, Mendel, Murlin, Noguchi, Shaffer, Teague, Terry, Torrey, Van Slyke, Wallace, Woodruff.

Members elected: Otto C. Glaser, Alfred G. Mayer, J. B. Murphy, Isaac Ott.

Twenty ninth meeting.

Cornell University Medical College, New York City. May 20, 1908. Silas P. Beebe in the chair.

Members present: Auer, Beebe, Carrel, Davenport, Elser, Ewing, Famulener, Foster, Gies, Harris, Hatcher, Janeway, Joseph, Kast, Levene, Levin, Loeb (L.), Meltzer, Murlin, Noguchi, Osborne, Salant, Wadsworth, Wallace, Wood.

Members elected: None.

REGISTER OF NAMES AND ADDRESSES OF THE MEMBERS OF THE SOCIETY FOR EXPERIMENTAL BIOLOGY AND MEDICINE.

ABBOTT, ALEXANDER C.....University of Pennsylvania.
ABEL, JOHN J.....Johns Hopkins University.
ADAMI, J. GEORGE.....McGill University, Montreal.
ADLER, ISAAC.....New York Polyclinic Medical School.
ALSBERG, CARL L.....Harvard University.
ATKINSON, JAMES P.....Department of Health, New York City.
AUER, JOHN.....Rockefeller Institute for Medical Research.

BANZHAF, EDWIN J.....Department of Health, New York City.
BARDEEN, CHARLES R.....University of Wisconsin.
BEEBE, SILAS P.....Cornell University Medical College.
BENEDICT, FRANCIS G....Nutrition Laboratory, Carnegie Institution, Boston.
BENSLEY, ROBERT R.....University of Chicago.
BERG, WILLIAM N.....U. S. Department of Agriculture, Washington, D. C.
BRINCKERHOFF, WALTER R.....U. S. Public Health and Marine-Hospital Service, Honolulu.
BROOKS, HARLOW.....New York University.
BUNTING, C. H.....University of Virginia.
BURTON-OPITZ, RUSSELL.....Columbia University.
BUXTON, B. H.....Cornell University Medical College.

CALKINS, GARY N.....Columbia University.
CANNON, WALTER B.....Harvard University.
CARLSON, A. J.....University of Chicago.
CARREL, ALEXIS.....Rockefeller Institute for Medical Research.
CHITTENDEN, R. H.....Yale University.
CLOWES, G. H. A.....University of Buffalo.
COLE, RUFUS I.....Johns Hopkins University.
CONKLIN, EDWIN G.....Princeton University.
COUNCILMAN, WILLIAM T.....Harvard University.
CRAMPTON, C. WARD.....Department of Education, New York City.
CRAMPTON, HENRY E. .....Columbia University.
CRILE, GEORGE W.....Western Reserve University, Cleveland.
CUNNINGHAM, RICHARD H.....Columbia University.
CUSHING, HARVEY W.....Johns Hopkins University.
CUSHNY, ARTHUR R.....University College, London.

DAKIN, H. D.....819 Madison Avenue, New York City.
DAVENPORT, CHARLES B.....Carnegie Institution's Station for Experimental Evolution, Cold Spring Harbor, Long Island, N. Y.
DONALDSON, H. H.....Wistar Institute of Anatomy, Philadelphia.
DUNHAM, EDWARD K.....New York University.
DUVAL, CHARLES W.....McGill University, Montreal.

EDSALL, DAVID L.....University of Pennsylvania.
ELSBERG, CHARLES A.....Mount Sinai Hospital.
ELSER, WILLIAM J.....Cornell University Medical College.
EMERSON, HAVEN.....Columbia University.
ERLANGER, JOSEPH.....University of Wisconsin.
EWING, JAMES.....Cornell University Medical College.

FAMULENER, L. W.....Department of Health, New York City.
FIELD, CYRUS W.....Department of Health, New York City.
FLEXNER, SIMON.....Rockefeller Institute for Medical Research.
FLOURNOY, THOMAS.....Bellevue Hospital, New York City.
FOLIN, OTTO.....Harvard University.
FORD, WILLIAM W.....Johns Hopkins University.
FOSTER, NELLIS B.....Columbia University.

GAGER, C. STUART.....University of Missouri.
GAY, FREDERICK P.....Harvard University.
GIBSON, ROBERT B.....University of Missouri.
GIES, WILLIAM J.....Columbia University.
GLASER, OTTO C.....University of Michigan.

HALSTED, WILLIAM S.....Johns Hopkins University.
HARRIS, ISAAC F.....449 E. 57th St., New York City.
HARRISON, ROSS G.....Yale University.
HATCHER, ROBERT A.....Cornell University Medical College.
HATAI, SHINKISHI.....Wistar Institute of Anatomy, Philadelphia.
HAWK, PHILIP B.....University of Illinois.
HEKTOEN, LUDVIG.....University of Chicago.
HENDERSON, YANDELL.....Yale University.
HERTER, CHRISTIAN A.....Columbia University.
HISS, PHILIP H.....Columbia University.
HOWELL, WILLIAM H.....Johns Hopkins University.
HUBER, CARL G.....University of Michigan.
HUNT, REID.....U. S. Public Health and Marine-Hospital Service, Hygienic Laboratory, Washington, D. C.

JACKSON, HOLMES C.....Albany Medical School.
JACOBS, WALTER A.....Rockefeller Institute for Medical Research.
JANEWAY, THEODORE C.....Sage Institute of Pathology, City Hospital, New York.
JOBLING, JAMES W.....Rockefeller Institute for Medical Research.

JONES, WALTER.....Johns Hopkins University.
JORDAN, EDWIN O.....University of Chicago.
JOSEPH, DON R.....Rockefeller Institute for Medical Research.

KAST, LUDWIG.....New York Postgraduate Medical School.
KASTLE, JOSEPH H.....U. S. Public Health and Marine-Hospital Service, Hygienic Laboratory, Washington, D. C.
KLOTZ, OSKAR.....McGill University, Montreal.
KOCH, WALDEMAR.....University of Chicago.

LEE, FREDERIC S.....Columbia University.
LEVENE, P. A.....Rockefeller Institute for Medical Research.
LEVIN, ISAAC.....Columbia University.
LEWIS, PAUL A.....Harvard University.
LILLIE, FRANK R.....University of Chicago.
LILLIE, RALPH S.....University of Pennsylvania.
LOEB, JACQUES.....University of California.
LOEB, LEO.....University of Pennsylvania.
LOEVENHART, ARTHUR S.....University of Wisconsin.
LOMBARD, WARREN P.....University of Michigan.
LUSK, GRAHAM.....New York University.

MACALLUM, A. B.....University of Toronto.
MACCALLUM, W. G.....Johns Hopkins University.
MACDOUGAL, D. T.....Carnegie Institution, Washington, D. C.
MACLEOD, J. J. R.....Western Reserve University, Cleveland.
MACNEAL, WARD J.....University of Illinois.
MALL, FRANKLIN P.....Johns Hopkins University.
MANDEL, ARTHUR R.....New York University.
MANDEL, JOHN A.....New York University.
MATHEWS, ALBERT P.....University of Chicago.
MAYER, ALFRED.....Marine Laboratory of the Carnegie Institution, Tortugas, Fla.
MELTZER, S. J.....Rockefeller Institute for Medical Research.
MENDEL, LAFAYETTE B.....Yale University.
MEYER, GUSTAVE M.....Columbia University.
MORGAN, THOMAS H.....Columbia University.
MURLIN, JOHN R.....New York University.
MURPHY, JOHN B.....Northwestern University Medical School, Chicago.
NOGUCHI, HIDEYO.....Rockefeller Institute for Medical Research.
NORRIS, CHARLES.....Bellevue Hospital, New York City.
NOVY, FREDERICK G.....University of Michigan.

OERTEL, HORST.....Sage Institute of Pathology, City Hospital, New York.
OPHÜLS, WILLIAM.....Cooper Medical College, San Francisco.
OPIE, EUGENE L.....Rockefeller Institute for Medical Research.

OSBORNE, THOMAS B.....Connecticut Agricultural Experiment Station, New Haven, Conn.
OTT, ISAAC..... Medico-Chirurgical College, Philadelphia.

PAPPENHEIMER, ALWIN M.....Bellevue Hospital, New York City.
PARK, WILLIAM H.....New York University.
PARKER, GEORGE H.....Harvard University.
PEARCE, RICHARD M.....New York University.
PFAFF, FRANZ.....Harvard University.
PORTER, WILLIAM T..... Harvard University.
PRATT, JOSEPH H.....Harvard University.

REICHERT, EDWARD T.....University of Pennsylvania.
RICHARDS, ALFRED N....Northwestern University Medical School, Chicago.
RICKETTS, HOWARD T.....University of Chicago.
ROBERTSON, T. BRAILSFORD.....University of California.
ROSENAU, MILTON J.....U. S. Public Health and Marine-Hospital Service, Hygienic Laboratory, Washington, D. C.

SALANT, WILLIAM.....U. S. Department of Agriculture, Washington, D. C.
SCHWYZER, FRITZ..... St. Francis Hospital, New York City.
SHAFFER, PHILIP A.....Cornell University Medical College.
SHERMAN, HENRY C.....Columbia University.
SIMON, CHARLES E.....Baltimore Medical College.
SMITH, THEOBALD..... Harvard University.
SOLLMANN, TORALD..... Western Reserve University, Cleveland.
STEWART, GEORGE N.....Western Reserve University, Cleveland.
STILES, PERCY G..... Massachusetts Institute of Technology.
STOOKEY, LYMAN B.....University of Southern California, Los Angeles.
STRONG, RICHARD P.....U. S. Public Health and Marine-Hospital Service, Manila.
SWEET, J. EDWIN.....University of Pennsylvania.
SYMMERS, DOUGLAS..... New York Hospital.

TAYLOR, ALONZO E..... University of California.
TEAGUE, OSCAR.....U. S. Department of the Interior, Bureau of Science, Manila.
TERRY, B. T..... Rockefeller Institute for Medical Research.
TORREY, JOHN C.....Cornell University Medical College.
TYZZER, E. E..... Harvard University.

UNDERHILL, FRANK P..... Yale University.

VAN SLYKE, DONALD D.....Rockefeller Institute for Medical Research.
VAUGHAN, VICTOR C..... University of Michigan.

WADSWORTH, AUGUSTUS B..... ..... Columbia University.
WALLACE, GEORGE B..... New York University.

WARTHIN, ALDRED S.....University of Michigan.
WEIL, RICHARD.....Cornell University Medical College.
WELCH, WILLIAM H.....Johns Hopkins University.
WELLS, H. GIDEON.....University of Chicago.
WILLIAMS, HERBERT U.....University of Buffalo.
WILSON, EDMUND B.....Columbia University.
WOLBACH, S. BURT.....Harvard University.
WOLF, CHARLES G. L.....Cornell University Medical College.
WOOD, FRANCIS C.....Columbia University.
WOODRUFF, LOSS LORANDE.....Yale University.

YATSU, NAOHIDÉ.....University of Japan.
YERKES, ROBERT M.....Harvard University.

Total number of members at the close of the academic year, 1907–'08: **162.**

OFFICERS.[1]

1903–1909.

	1903–'04	1904–'05	1905–'06	1906–'07	1907–'08	1908–'09
President	Meltzer	Meltzer	Wilson	Flexner	Flexner	Lee
Vice-President	Park	Ewing	Dunham	Dunham	Morgan	Morgan
Librarian	Lusk	Lusk	Lusk	———	———	———
Treasurer	Calkins	Calkins	Calkins	Calkins	Calkins	Lusk
Secretary	Gies	Gies	Gies	Gies	Gies	Gies

[1] Council — The Past Presidents and the Officers.

CLASSIFIED LIST OF MEMBERS OF THE SOCIETY FOR EXPERIMENTAL BIOLOGY AND MEDICINE.

Resident (Greater New York).

Bellevue Hospital.—Thomas Flournoy, Charles Norris, Alwin M. Pappenheimer.

Columbia University.—Russell Burton-Opitz, Gary N. Calkins, Henry E. Crampton, Richard H. Cunningham, Haven Emerson, Nellis B. Foster, William J. Gies, Christian A. Herter, Philip H. Hiss, Frederic S. Lee, Isaac Levin, Gustave M. Meyer, Thomas H. Morgan, Henry C. Sherman, Augustus B. Wadsworth, Edmund B. Wilson, Francis C. Wood.

Cornell University Medical College.—S. P. Beebe, B. H. Buxton, William J. Elser, James Ewing, Robert A. Hatcher, Philip A. Shaffer, John C. Torrey, Richard Weil, C. G. L. Wolf.

Mt. Sinai Hospital.—Charles A. Elsberg.

New York Department of Education.—C. Ward Crampton.

New York Department of Health.—James P. Atkinson, Edwin J. Banzhaf, L. W. Famulener, Cyrus W. Field.

New York Hospital.—Douglas Symmers.

New York Polyclinic Medical School.—Isaac Adler.

New York Post-Graduate Medical School.—Ludwig Kast.

New York University.—Harlow Brooks, Edward K. Dunham, Graham Lusk, Arthur R. Mandel, John A. Mandel, John R. Murlin, William H. Park, Richard M. Pearce, George B. Wallace.

Rockefeller Institute for Medical Research.—John Auer, Alexis Carrel, Simon Flexner, Walter A. Jacobs, James W. Jobling, Don R. Joseph, P. A. Levene, S. J. Meltzer, Hideyo Noguchi, Eugene L. Opie, B. T. Terry, Donald D. Van Slyke.

Sage Institute of Pathology, City Hospital.—Theodore C. Janeway, Horst Oertel.

St. Francis Hospital.—Fritz Schwyzer.

---

819 Madison Avenue.—H. D. Dakin.

449 E. 57th Street.—Isaac F. Harris.

Non-Resident.

Albany Medical College.—Holmes C. Jackson.

Baltimore Medical College.—Charles E. Simon.

Carnegie Institution of Washington.—Francis G. Benedict (*Nutrition Laboratory, Boston*), Charles B. Davenport (*Station for Experimental Evolution, Cold Spring Harbor, N. Y.*), D. T. MacDougal (*Washington*), Alfred G. Mayer (*Marine Laboratory, Tortugas, Fla.*).

Connecticut Agricultural Experiment Station (New Haven).—Thomas B. Osborne.

Cooper Medical College (San Francisco).—William Ophüls.

Harvard University.—Carl L. Alsberg, Walter B. Cannon, W. T. Councilman, Otto Folin, Frederick P. Gay, Paul A. Lewis, G. H. Parker, Franz Pfaff, W. T. Porter, Joseph H. Pratt, Theobald Smith, E. E. Tyzzer, S. Burt Wolbach, Robert M. Yerkes.

Johns Hopkins University.—John J. Abel, Rufus I. Cole, Harvey W. Cushing, W. W. Ford, W. S. Halsted, William H. Howell, Walter Jones, W. G. MacCallum, F. P. Mall, William H. Welch.

Massachusetts Institute of Technology.—Percy G. Stiles.

McGill University (Montreal).—J. George Adami, Charles W. Duval, Oskar Klotz.

Medico-Chirurgical College (Philadelphia).—Isaac Ott.

Northwestern University Medical School (Chicago).—J. B. Murphy, Alfred N. Richards.

Princeton University.—Edwin G. Conklin.

U. S. Department of Agriculture (Washington, D. C.).—Wm. N. Berg, William Salant.

U. S. Department of the Interior, Bureau of Science, Manila.—Oscar Teague.

U. S. Public Health and Marine Hospital Service.—Walter R. Brinckerhoff (*Honolulu*), Reid Hunt (*Washington*), J. H. Kastle (*Washington*), M. J. Rosenau (*Washington*), Richard P. Strong (*Manila*).

University College (London).—Arthur R. Cushny.

University of Buffalo.—G. H. A. Clowes, Herbert U. Williams.

University of California.—Jacques Loeb, T. Brailsford Robertson, Alonzo E. Taylor.

University of Chicago.—R. R. Bensley, A. J. Carlson, Ludvig Hektoen, Edwin O. Jordan, Waldemar Koch, Frank R. Lillie, Albert P. Mathews, H. T. Ricketts, H. Gideon Wells.

University of Illinois.—Philip B. Hawk, Ward J. MacNeal.

University of Japan.—Naohidé Yatsu.

University of Michigan.—Otto C. Glaser, Carl G. Huber, Warren P. Lombard, Frederick G. Novy, Victor C. Vaughan, Aldred S. Warthin.

University of Missouri.—C. Stuart Gager, Robert B. Gibson.

University of Pennsylvania.—Alexander C. Abbott, David L. Edsall, Ralph S. Lillie, Leo Loeb, Edward T. Reichert, J. Edwin Sweet.

University of Southern California (Los Angeles).—Lyman B. Stookey.

University of Toronto.—A. B. Macallum.

University of Virginia.—C. H. Bunting.

University of Wisconsin.—Charles R. Bardeen, Joseph Erlanger, Arthur S. Loevenhart.

Western Reserve University (Cleveland).—George W. Crile, J. J. R. Macleod, Torald Sollmann, G. N. Stewart.

Wistar Institute of Anatomy (Philadelphia).—H. H. Donaldson, Shinkishi Hatai.

Yale University.—R. H. Chittenden, Ross G. Harrison, Yandell Henderson, Lafayette B. Mendel, Frank P. Underhill, L. L. Woodruff.

INDEX

OF THE

SCIENTIFIC PROCEEDINGS.

By EMILY C. SEAMAN.

[THE NUMERALS IN THIS INDEX CORRESPOND WITH THE NUMERALS IN PARENTHESIS ABOVE THE TITLES OF THE ABSTRACTS. PAGES ARE NOT INDICATED.]

Acid. See carnaubic, hydrochloric, organic, oxalic, uric.

Adrenalin, 279 : effect on circulation through the kidneys.

Age: physiological, 271 ; plasticity in the dancing mouse, 292.

Alcohol. See protozoa.

Ammonia, 338 : determination by the Folin method in urines containing crystalline ammonio-magnesium phosphate.

Ammonio-magnesium phosphate, 338. See ammonia.

Anaphylaxis, 291 ; specificity, 302 ; to horse serum, 313.

Anesthesia. See ether.

Anti-bodies, 289 ; formation.

Antidiphtheric serum, 269 : relative value of antitoxin.

Antitoxin, 269 : value of, in antidiphtheric serum.

Apparatus. See viscosimeter.

Arabinose, 332 : glycogen formation in chicks.

Arterial pressure, 257 : cause of cardiac insufficiency.

Arterial segments, 322 : transplantation.

Arteriosclerosis, 282.

Artery. See carotid, popliteal.

Atoxyl, 308 : prevention of syphilis in Macacus Rhesus.

Auricles. See nervous coördination.

Autolysis: influence of iodides, 328 ; relation of thyroids, 329.

Bardach's protein test, 335.

Bladder, 279 : relation of renal blood flow to pressure in the ureter and bladder.

Blood: *flow* — relation of renal blood flow to pressure in the ureter and bladder, 279 ; *vessels* — innervation in the intestine, 312, in the kidney, 311, transplantation, 322, 324. See arterial pressure, carotid artery, circulation, coagulation time, corpuscle resistance, hemolytic reactions, horse serum, isohemagglutination, polycythemia.

Body, 299 : influence of diet on chemical composition.

Body weight, 326 : relation to weight of stomach- and cecum-contents.

Brain. See medulla oblongata.

Calcium: action upon the pupil and its relation to the effects of mydriatics, 305 ; calcium and magnesium salts upon development of rigor mortis, 263 ; antagonistic action upon the inhibitory effect of magnesium, 274 ; relation of calcium metabolism to tetany and the cure

[THE NUMERALS CORRESPOND WITH THOSE ABOVE THE ABSTRACTS.]

of tetany by administration of calcium, 303; relative toxicity, 304.

Calcium metabolism, 303: relation to tetany.

Carbohydrate, 296: influence on protein metabolism of a fasting pregnant dog. See arabinose.

Cardiac insufficiency, 257: due to high arterial pressure.

Carnaubic acid, 288: from beef kidneys.

Carotid artery, 266: depressor reaction obtainable by traction.

Cecum contents, 326: relation of weight to body weight.

Cells, 270: effect of light in fluorescent solution after addition of potassium cyanide.

Chemical composition: influence of diet on the composition of the body, 299; of nonstriated muscle, 300.

Chromosomes, 285.

Circulation: portal, 280; renal, 279.

Coagulation time, 276: relation of the coagulation time of the blood to thrombosis in phlebitis.

Cold, 295: influence in phlorhizin glycosuria

Colloids, 290: precipitation by sera.

Coördination, 298: auricles and ventricle of the lizard heart.

Corpuscle resistance, 289: in blood of immunized animals.

Crystallography, 293: hemoglobins.

Cyanide. See potassium cyanide.

Dancing mouse, 292: plasticity to age and sex.

Depressor reaction, 266: obtainable by traction on carotid artery.

Diet, 299: influence on the chemical composition of the body.

Digestion. See gastric.

Distribution, 287: *Partula* in Polynesia.

Eserin, 327: inhibitory influence of magnesium on its effects.

Ether anesthesia, 268: on excretion of nitrogen.

Excretion (nitrogen), 268: influence of ether anesthesia. See metabolism.

Exercise, 295: influence in phlorhizin glycosuria.

Fasting pregnant dog, 296: influence of carbohydrate on the protein metabolism.

"Female producing" spermatozoa, 286: production in phylloxerans.

Fever, 323: unknown origin; nitrogen metabolism.

Fibers. See glycogenolytic.

Fluorescent solution. See cells.

Folin method (ammonia), 338: inefficient for urines containing crystalline ammonio-magnesium phosphate.

Function, 265: reëstablishment in transplanted kidneys.

Gastric: *digestion* — hydrochloric acid secretion, 319; *hormon* — in the salivary glands, 325; *juice* — effects of organic acids on secretion, 320; *peristalsis* — after section of the vagi and splanchnic nerves, 272; *secretion* — effect of mechanical obstruction of the pylorus, 321.

Geographical distribution, 287: *Partula* in Polynesia.

Germicidal, 294: property of milk.

Glycogen formation, 332: from arabinose in chicks.

Glycogenolytic fibers, 307: in the greater splanchnic nerves.

Glycosuria, 295: influence of cold and exercise in phlorhizin glycosuria.

Growth, 264. See tumor.

Guinea-pig, 260: hypersensitiveness to horse serum.

[THE NUMERALS CORRESPOND WITH THOSE ABOVE THE ABSTRACTS.]

Heart, 273 : effect of stimulation of vagi upon development of rigor mortis of the mammalian heart. See nervous coördination.

Heloderma suspectum, 317 : hemolytic action of venom.

Hemin, 267 : Teichmann's method for obtaining crystals.

Hemoglobins, 293 : crystallography.

Hemolytic action, 317 : venom of *Heloderma suspectum.*

Hemolytic reactions, 278 : of the blood in dogs with transplantable lymphosarcoma.

Hepatic uricolysis, 333 : oxalic acid as a product.

Heredity, 315 : human physical characteristics.

Hormon, 325 : gastric hormon in salivary glands.

Horse serum: hypersensitiveness of guinea-pigs, 260 ; anaphylaxis to horse serum, 313.

Hydrochloric acid, 319 : secretion during gastric digestion.

Hypersensitiveness, 260 : guinea-pig to horse serum.

Immunity. See antitoxin, corpuscle resistance, hypersensitiveness.

Implantation. See tissue.

Inhibition, 274 : antagonistic action of calcium on that caused by magnesium.

Innervation of blood vessels: renal, 311 ; intestinal, 312.

Intestine, 312: innervation of the blood vessels.

Iodides: influence on autolysis, 328 ; pharmacology, 331.

Isohemagglutination, 262 : rôle of tonicity.

Kidneys: circulation, 279 ; innervation of the blood vessels, 311 ; isolation of carnaubic acid, 288 ; reestablishment of function, 265.

Life cycle, 334: *Paramecium.*

Light, 270 : effect on cells in fluorescent solution after addition of potassium cyanide.

Liver. See hepatic uricolysis.

Lizard. See nervous coördination.

Lung, 259: pneumothorax and posture.

Macacus Rhesus, 308 : prevention of syphilis by atoxyl.

Magnesium : antagonistic action of calcium upon the inhibitory effect of magnesium, 274 ; magnesium and calcium salts upon development of rigor mortis, 263 ; inhibitory influence on effects of eserin, 327 ; metabolic effects, 337 ; relative toxicity, 304.

Medulla oblongata, 283 : influence of substances, applied directly, on respiratory rhythm.

Metabolism : in experimental polycythemia, 336 ; affected by magnesium sulfate, 337. See calcium, excretion of nitrogen, nitrogen, protein metabolism.

Metaplasia, 284 : rat tumor.

Metastasis, 284 : rat tumor.

Methods : ammonia determination in urine containing ammonio-magnesium phosphate (Folin's), 338 ; experimental surgery, 265, 275, 277, 297, 316, 322 ; new protein test (Bardach's), 335 ; preparation of carnaubic acid from beef kidneys (Dunham's), 288 ; preparation of protagon (Wilson and Cramer's), 314 ; production of hemin crystals (Atkinson and Kendall's), 267.

Milk, 294 : germicidal property.

Monkey. See Macacus Rhesus.

[THE NUMERALS CORRESPOND WITH THOSE ABOVE THE ABSTRACTS.]

Mouse, 261 : sporozoan in peptic glands.
Muscle. See rigor mortis.
Mydriatics. See calcium.

Nerves. See vagi and splanchnic nerves.
Nervous coördination, 298 : of auricles and ventricle of the lizard heart.
Nitrogen, 268 : excretion under influence of ether anesthesia.
Nitrogen metabolism, 323 : in a case presenting short paroxysms of fever.
Nitrogenous constituents (urine), 337 ; influence of magnesium sulfate on their partition.
Nonstriated muscle, 300 : chemical composition.
Nucleic acids, 310.

Organic acids, 320 : effect on secretion of gastric juice.
Oxalic acid, 333 : in hepatic uricolysis.

Paramecium, 334 : life cycle.
Parasite, 261. See sporozoan.
Parathyroid glands, 297 : transplantation in dogs.
Partula, 287: distribution in Polynesia.
Pentose. See arabinose.
Peptic glands, 261 : sporozoan.
Peristalsis. See gastric.
Phlebitis, 276 : relation of coagulation time of the blood to thrombosis in phlebitis.
Phlorhizin glycosuria, 295 : influence of cold and exercise.
Placenta, 316 : experimental production of maternal part.
Plasticity, 292 : sex and age in the dancing mouse.
Pneumothorax, 259 : and posture.
Poison, 301 : susceptibility of protozoa to poison due to treatment with alcohol.
Polycythemia, 336 : metabolism.
Polynesia, 287 : distribution of *Partula*.
Popliteal artery, 275 : transplantation of a segment from a man to a bitch.
Portal circulation, 280.
Posture, 259 : and pneumothorax.
Potassium, 304 : relative toxicity.
Potassium cyanide, 270 : effect of light on cells in fluorescent solution after addition of potassium cyanide.
Precipitation, 290 : inorganic colloids by sera.
Pregnant dog, 296 : influence of carbohydrate on the protein metabolism.
Pressure. See blood flow.
Protagon, 314 : prepared by the Wilson-Cramer method.
Protein metabolism, 296 : influence of carbohydrate.
Proteins, 318 : from seeds ; biological relations.
Protein test, 335 : Bardach's.
Protozoa, 301 : susceptibility to poison due to treatment with alcohol.
Pupil, 305 : action of calcium.
Pylorus, 321 : effect of mechanical obstruction on gastric secretion.

Rat tumor. See tumor.
Reactive power, 277 : of the white rat to tissue implantation.
Remote result, 275 : of the transplantation of a segment of popliteal artery from a man to a bitch.
Respiratory rhythm, 283 : influence of various substances applied directly to the medulla oblongata.
Rigor mortis : development of, 273 ; effect of calcium and magnesium salts, 263.

Salivary glands, 325 : gastric hormon.
Secretion : effects of organic acids on

[THE NUMERALS CORRESPOND WITH THOSE ABOVE THE ABSTRACTS.]

secretion of gastric juice, 320; hydrochloric acid during gastric digestion, 319. See gastric.

Sera, 290: cause of precipitation of inorganic colloids.

Serum, 269: value of antitoxin in antidiphtheric serum. See horse serum.

Sex, 292: plasticity in dancing mouse.

Sodium, 304: relative toxicity.

Specificity, 302: of anaphylaxis.

Spermatozoa, 286: production of two kinds in phylloxerans.

Splanchnic, 272: gastric peristalsis after section of vagi and splanchnic nerves.

Splanchnic nerves, 307: nature of the so-called glycogenolytic fibers.

Sporozoan, 261: found in peptic glands of common mouse.

Stimulation, 273: of the vagi upon development of rigor mortis of the mammalian heart.

Stomach. See gastric.

Stomach contents, 326: relation of weight to body weight.

Strophanthin, 306: destruction in the animal organism.

Sugar. See arabinose.

Susceptibility, 301: of protozoa to poison due to treatment with alcohol.

Syphilis, 308: prevention in Macacus Rhesus by atoxyl.

Teichmann's method, 267: for obtaining hemin crystals.

Test, 335: Bardach's protein test.

Tetany, 303: relation of calcium metabolism and cure of tetany by administration of calcium.

Thrombosis, 276: relation of the coagulation time of the blood to thrombosis in phlebitis.

Thyroid glands: physiology, 330; relation to autolysis, 329.

Tissue implantation, 277: reactive power of the white rat.

Tonicity, 262: rôle in human isohemagglutination.

Toxicity, 304: of the chlorides of magnesium, calcium, potassium and sodium.

Traction, 266: on carotid artery, cause of depressor reaction.

Transplantable lymphosarcoma, 278: hemolytic reactions of the blood in dogs.

Transplantation: blood vessels, 324; devitalized arterial segments, 322; parathyroid glands in dogs, 297; segments of popliteal artery from a man to a bitch, 275.

Tumor: metaplasia and metastasis, 284; notes on a rat tumor, 309; restraint and promotion of growth, 264.

Ureter, 279: relation of renal blood flow to pressure in the ureter and bladder.

Uric acid. See uricolysis.

Uricolysis, 333: oxalic acid as a product of hepatic uricolysis.

Urine. See ammonia, nitrogenous constituents.

Vagi: effect of stimulation upon the development of rigor mortis of the mammalian heart, 273; gastric peristalsis after section of vagi and splanchnic nerves, 272.

Vasomotor reactions, 279: renal.

Venom, 317: hemolytic action.

Ventricle. See nervous coördination.

Viscosimeter, 281: Burton-Opitz form.

Wilson-Cramer method, 314: study of "protagon" prepared by it.

Herbert D. Jordan

PROCEEDINGS

OF THE

SOCIETY FOR

EXPERIMENTAL BIOLOGY AND MEDICINE

TWENTY FIFTH MEETING

COLLEGE OF PHYSICIANS AND SURGEONS

COLUMBIA UNIVERSITY

NEW YORK CITY

OCTOBER 16, 1907

VOLUME V

No. 1

NEW YORK

NOVEMBER 15, 1907

CONTENTS.

HAVEN EMERSON: Cardiac insufficiency due to high arterial pressure. 1 (257).

GEORGE B. WALLACE and A. N. RICHARDS: Effect of potassium cyanide upon metabolism. 2 (258).

CHARLES A. ELSBERG: Pneumothorax and posture. 3 (259).

PAUL A. LEWIS: The hypersensitiveness of the guinea pig to horse serum. 4 (260).

E. E. TYZZER: A sporozoan found in the peptic glands of the common mouse. 5 (261).

FREDERICK P. GAY: The rôle of tonicity in human isohemagglutination. 6 (262).

S. J. MELTZER and JOHN AUER: Effects of calcium and magnesium salts upon the development of rigor mortis. 7 (263).

SIMON FLEXNER and JAMES W. JOBLING: Restraint and promotion of tumor growth. 8 (264).

ALEXIS CARREL: Reestablishment of function in transplanted kidneys. 9 (265).

TORALD SOLLMANN and E. D. BROWN: A depressor reaction obtainable by traction on the carotid artery. 10 (266).

JAMES P. ATKINSON and ARTHUR I. KENDALL: A modification of Teichmann's method for obtaining hemin crystals, with a demonstration of specimens. 11 (267).

PHILIP B. HAWK: The influence of ether anesthesia on the excretion of nitrogen by dogs. 12 (268).

EDNA STEINHARDT and EDWIN J. BANZHAF: The relative value of antitoxin and other curative substances in antidiphtheric serum. 13 (269).

The proceedings of the Society for Experimental Biology and Medicine are published as soon as possible after each meeting. Regular meetings of the Society are held in New York on the third Wednesdays of October, December, February, March, April, May and June. A volume of the proceedings consists of the numbers issued during an academic year.

The price per volume, sent postage prepaid, is one dollar. The price of copies of the proceedings of any meeting is fifteen cents each, postage prepaid. Subscriptions are payable in advance.

Address communications to any of the following members of the Council of the Society:

PAST PRESIDENTS — S. J. Meltzer, Rockefeller Institute for Medical Research; Edmund B. Wilson, Columbia University.

PRESIDENT — Simon Flexner, Rockefeller Institute for Medical Research.

VICE-PRESIDENT — Thomas H. Morgan, Columbia University.

TREASURER — Gary N. Calkins, Columbia University.

SECRETARY — William J. Gies, Columbia University.

MANAGING EDITOR — The Secretary, College of Physicians and Surgeons, 437 W. 59th St., New York City.

PROCEEDINGS

OF THE

SOCIETY FOR

EXPERIMENTAL BIOLOGY AND MEDICINE

TWENTY SIXTH MEETING

ROCKEFELLER INSTITUTE FOR MEDICAL RESEARCH

NEW YORK CITY

DECEMBER 18, 1907

VOLUME V

No. 2

NEW YORK

JANUARY 15, 1908

CONTENTS.

ELIZABETH COOKE and LEO LOEB: The effect of light on cells in fluorescent solutions after addition of potassium cyanide. 14 (270).

C. WARD CRAMPTON: Physiological age. 15 (271).

JOHN AUER: Gastric peristalsis after section of the vagi and splanchnic nerves. 16 (272).

DON R. JOSEPH and S. J. MELTZER: The effect of stimulation of the vagus nerves upon the development of rigor mortis of the mammalian heart. 17 (273).

S. J. MELTZER and JOHN AUER: The antagonistic action of calcium upon the inhibitory effect of magnesium. 18 (274).

ALEXIS CARREL: Remote result of the transplantation of a segment of popliteal artery from a man to a bitch. 19 (275).

HARLOW BROOKS and B. C. CROWELL: Concerning the relation of the coagulation time of the blood to thrombosis in phlebitis. 20 (276).

ISAAC LEVIN: The reactive power of the white rat to tissue implantation. Second communication. 21 (277).

RICHARD WEIL: The hemolytic reactions of the blood in dogs with transplantable lymphosarcoma. 22 (278).

R. BURTON-OPITZ and D. R. LUCAS: On the circulation through the kidneys. I. On vaso-motor reactions. II. The renal blood flow in relation to the pressure in the ureter and bladder. III. The effect of solutions of adrenalin. 23 (279).

R. BURTON-OPITZ: Some data regarding the portal circulation. 24 (280).

R. BURTON-OPITZ: A clinical viscosimeter. 25 (281).

ISAAC ADLER and O. HENSEL: Studies in experimental arteriosclerosis. 26 (282).

T. BRAILSFORD ROBERTSON: On the influence of various substances, applied directly to the medulla oblongata, upon the respiratory rhythm in frogs. 27 (283).

SIMON FLEXNER and J. W. JOBLING: Metaplasia and metastasis of a rat tumor. 28 (284).

The proceedings of the Society for Experimental Biology and Medicine are published as soon as possible after each meeting. Regular meetings of the Society are held in New York on the third Wednesdays of October, December, February, April and May. A volume of the proceedings consists of the numbers issued during an academic year.

The price per volume, sent postage prepaid, is one dollar. The price of copies of the proceedings of any meeting is fifteen cents each, postage prepaid. Subscriptions are payable in advance.

Address communications to any of the following members of the Council of the Society:

PAST PRESIDENTS — S. J. Meltzer, Rockefeller Institute for Medical Research; Edmund B. Wilson, Columbia University.

PRESIDENT — Simon Flexner, Rockefeller Institute for Medical Research.

VICE-PRESIDENT — Thomas H. Morgan, Columbia University.

TREASURER — Gary N. Calkins, Columbia University.

SECRETARY — William J. Gies, Columbia University.

MANAGING EDITOR — The Secretary, College of Physicians and Surgeons, 437 W. 59th St., New York City.

PROCEEDINGS

Edwin O. Jordan

OF THE

SOCIETY FOR

EXPERIMENTAL BIOLOGY AND MEDICINE

TWENTY SEVENTH MEETING

SCHERMERHORN HALL, COLUMBIA UNIVERSITY

NEW YORK CITY

FEBRUARY 19, 1908

VOLUME V

No. 3

NEW YORK

MARCH 15, 1908

CONTENTS.

EDMUND B. WILSON: An exhibition of photographs of chromosomes, with explanatory comment. 29 (285).

T. H. MORGAN: The production of two kinds of spermatozoa in phylloxerans — functional "female producing" and rudimentary spermatozoa. 30 (286).

HENRY E. CRAMPTON: Physiological problems of the geographical distribution of *Partula* in Polynesia, with demonstration of specimens. 31 (287).

EDWARD K. DUNHAM: Note on the isolation of carnaubic acid from beef kidneys. 32 (288).

FREDERICK P. GAY: The change of corpuscle resistance in the blood of immunized animals, coincident with the formation of anti-bodies. 33 (289).

CYRUS W. FIELD: Further observations on the precipitation of inorganic colloids by sera. 34 (290).

EDWIN J. BANZHAF and L. W. FAMULENER: A note on anaphylaxis. 35 (291).

ROBERT M. YERKES: The relation of plasticity to sex and age in the dancing mouse. 36 (292).

EDWARD T. REICHERT and AMOS P. BROWN: The crystallography of hemoglobins. 37 (293).

M. J. ROSENAU and GEO. W. MCCOY: The germicidal property of milk. 38 (294).

The proceedings of the Society for Experimental Biology and Medicine are published as soon as possible after each meeting. Regular meetings of the Society are held in New York on the third Wednesdays of October, December, February, April and May. A volume of the proceedings consists of the numbers issued during an academic year.

The price per volume, sent postage prepaid, is one dollar. The price of copies of the proceedings of any meeting is twenty cents each, postage prepaid. Subscriptions are payable in advance.

Address communications to any of the following members of the Council of the Society:

PAST PRESIDENTS — S. J. Meltzer, Rockefeller Institute for Medical Research; Edmund B. Wilson, Columbia University; Simon Flexner, Rockefeller Institute for Medical Research.

PRESIDENT — Frederic S. Lee, Columbia University.

VICE-PRESIDENT — Thomas H. Morgan, Columbia University.

TREASURER — Graham Lusk, New York University.

SECRETARY — William J. Gies, Columbia University.

MANAGING EDITOR — The Secretary, College of Physicians and Surgeons, 437 W. 59th St., New York City.

CLASSIFIED LIST OF MEMBERS OF THE SOCIETY FOR EXPERIMENTAL BIOLOGY AND MEDICINE.

Resident (Greater New York).

Bellevue Hospital.—Thomas Flournoy, Charles Norris, Alwin M. Pappenheimer.

Columbia University.—Russell Burton-Opitz, Gary N. Calkins, Henry E. Crampton, Richard H. Cunningham, Haven Emerson, Nellis B. Foster, William J. Gies, Christian A. Herter, Philip H. Hiss, Frederic S. Lee, Isaac Levin, T. H. Morgan, Alfred N. Richards, H. C. Sherman, Augustus B. Wadsworth, Edmund B. Wilson, Francis C. Wood.

Cornell University Medical College.—S. P. Beebe, B. H. Buxton, William J. Elser, James Ewing, Robert A. Hatcher, Philip A. Shaffer, Oscar Teague, John C. Torrey, Richard Weil, C. G. L. Wolf.

Mt. Sinai Hospital.—Charles A. Elsberg.

New York Botanical Garden.—C. Stuart Gager.

New York Department of Education.—C. Ward Crampton.

New York Department of Health.—James P. Atkinson, Edwin J. Banzhaf, L. W. Famulener, Cyrus W. Field.

New York Hospital.—Douglas Symmers.

New York Polyclinic Medical School.—Isaac Adler.

New York Post-Graduate Medical School.—Ludwig Kast.

New York University.—Harlow Brooks, Edward K. Dunham, Graham Lusk, Arthur R. Mandel, John A. Mandel, John R. Murlin, William H. Park, George B. Wallace.

Rockefeller Institute for Medical Research.—John Auer, Alexis Carrel, Simon Flexner, Walter A. Jacobs, James W. Jobling, Don R. Joseph, P. A. Levene, S. J. Meltzer, Hideyo Noguchi, Eugene L. Opie, B. T. Terry, Donald D. Van Slyke.

Sage Institute of Pathology, City Hospital.—Theodore C. Janeway, Horst Oertel.

St. Francis Hospital.—Fritz Schwyzer.

---

819 Madison Avenue.—H. D. Dakin.

19 W. 42d Street.—Isaac F. Harris.

Non-Resident.

Albany Medical College.—Holmes C. Jackson, Richard M. Pearce.

Baltimore Medical College.—Charles E. Simon.

Carnegie Institution of Washington.—Francis G. Benedict (*Nutrition Laboratory, Boston*), Charles B. Davenport (*Station for Experimental Evolution, Cold Spring Harbor, N. Y.*), D. T. MacDougal (*Washington*).

Connecticut Agricultural Experiment Station (New Haven).—Thomas B. Osborne.

Cooper Medical College (San Francisco).—William Ophüls.

Harvard University.—Carl L. Alsberg, Walter B. Cannon, W. T. Councilman, Otto Folin, Frederick P. Gay, Paul A. Lewis, G. H. Parker, Franz Pfaff, W. T. Porter, Joseph H. Pratt, Theobald Smith, E. E. Tyzzer, S. Burt Wolbach, Robert M. Yerkes.

Johns Hopkins University.—John J. Abel, Rufus I. Cole, Harvey W. Cushing, W. W. Ford, W. S. Halsted, William H. Howell, Walter Jones, A. S. Loevenhart, W. G. MacCallum, F. P. Mall, William H. Welch.

Massachusetts Institute of Technology.—Percy G. Stiles.

McGill University (Montreal).—J. George Adami, Charles W. Duval, Oskar Klotz.

U. S. Department of Agriculture (Washington, D. C.).—Wm. N. Berg.

U. S. Public Health and Marine Hospital Service.—Walter R. Brinckerhoff (*Honolulu*), Reid Hunt (*Washington*), J. H. Kastle (*Washington*), M. J. Rosenau (*Washington*), Richard P. Strong (*Manila*).

University College (London).—Arthur R. Cushny.

University of Alabama.—William Salant.

University of Buffalo.—G. H. A. Clowes, Herbert U. Williams.

University of California.—Jacques Loeb, T. Brailsford Robertson, Alonzo E. Taylor.

University of Chicago.—R. R. Bensley, A. J. Carlson, Ludvig Hektoen, Edwin O. Jordan, Waldemar Koch, Frank R. Lillie, Albert P. Mathews, H. T. Ricketts, H. Gideon Wells.

University of Illinois.—Philip B. Hawk, Ward J. MacNeal.

University of Japan.—Naohidé Yatsu.

University of Michigan.—Carl G. Huber, Warren P. Lombard, Frederick G. Novy, Victor C. Vaughan, Aldred S. Warthin.

University of Missouri.—Robert B. Gibson.

University of Pennsylvania.—Alexander C. Abbott, E. G. Conklin, David L. Edsall, Ralph S. Lillie, Leo Loeb, Edward T. Reichert, J. Edwin Sweet.

University of Southern California (Los Angeles).—Lyman B. Stookey.

University of Syracuse.—Gustave M. Meyer.

University of Toronto.—A. B. Macallum.

University of Virginia.—C. H. Bunting.

University of Wisconsin.—Charles R. Bardeen, Joseph Erlanger.

Western Reserve University (Cleveland).—George W. Crile, J. J. R. Macleod, Torald Sollmann, G. N. Stewart.

Wistar Institute of Anatomy (Philadelphia).—H. H. Donaldson, Shinkishi Hatai.

Yale University.—R. H. Chittenden, Ross G. Harrison, Yandell Henderson, Lafayette B. Mendel, Frank P. Underhill, L. L. Woodruff.

Members present at the twenty seventh meeting:

Auer, Banzhaf, Crampton (H. E.), Dunham, Famulener, Gager, Gay, Gies, Joseph, Kast, Lusk, Meltzer, Morgan, Murlin, Sherman, Teague, Wilson.

Members elected at the twenty seventh meeting:

L. W. Famulener, C. Stuart Gager, W. S. Halsted, Walter A. Jacobs, Theodore C. Janeway, Don R. Joseph, Frank R. Lillie, Alwin M. Pappenheimer, Donald D. Van Slyke.

Officers elected at the twenty seventh meeting:

President, Frederic S. Lee; *Vice-president*, Thomas H. Morgan; *Treasurer*, Graham Lusk; *Secretary*, William J. Gies.

Dates of the next two regular meetings:

April 15, 1908.
May 20, 1908.

CLASSIFIED LIST OF MEMBERS OF THE SOCIETY FOR EXPERIMENTAL BIOLOGY AND MEDICINE.

Resident (Greater New York).

Bellevue Hospital.—Thomas Flournoy, Charles Norris.

Columbia University.—William N. Berg, Russell Burton-Opitz, Gary N. Calkins, Henry E. Crampton, Richard H. Cunningham, Haven Emerson, Nellis B. Foster, William J. Gies, Christian A. Herter, Philip H. Hiss, Frederic S. Lee, T. H. Morgan, Horst Oertel, Alfred N. Richards, H. C. Sherman, Augustus B. Wadsworth, Edmund B. Wilson, Francis C. Wood.

Cornell University Medical College.—S. P. Beebe, B. H. Buxton, William J. Elser, James Ewing, Robert A. Hatcher, Philip A. Shaffer, Oscar Teague, John C. Torrey, Richard Weil, C. G. L. Wolf.

Mt. Sinai Hospital.—Charles A. Elsberg.

New York Department of Education.—C. Ward Crampton.

New York Department of Health.—James P. Atkinson, Edwin J. Banzhaf, Cyrus W. Field.

New York Hospital.—Douglas Symmers.

New York Polyclinic Medical School.—Isaac Adler.

New York University.—Harlow Brooks, Edward K. Dunham, Graham Lusk, Arthur R. Mandel, John A. Mandel, John R. Murlin, William H. Park, George B. Wallace.

Rockefeller Institute for Medical Research.—John Auer, Alexis Carrel, Simon Flexner, James W. Jobling, P. A. Levene, S. J. Meltzer, Hideyo Noguchi, Eugene L. Opie, B. T. Terry.

St. Francis Hospital.—Fritz Schwyzer.

Sydenham Hospital.—Isaac Levin.

---

819 Madison Avenue.—H. D. Dakin.

19 W. 42d Street.—Isaac F. Harris.

43 W. 52d Street.—Ludwig Kast.

Non-Resident.

Albany Medical College.—Holmes C. Jackson, Richard M. Pearce.

Baltimore Medical College.—Charles E. Simon.

Carnegie Institution of Washington.—Francis G. Benedict (*Nutrition Laboratory, Boston*), Charles B. Davenport (*Station for Experimental Evolution, Cold Spring Harbor, N. Y.*), D. T. MacDougal (*Washington*).

Connecticut Agricultural Experiment Station (New Haven).—Thomas B. Osborne.

Cooper Medical College (San Francisco).—William Ophüls.

Harvard University.—Carl L. Alsberg, Walter B. Cannon, W. T. Councilman, Otto Folin, Frederick P. Gay, Paul A. Lewis, G. H. Parker, Franz Pfaff, W. T. Porter, Joseph H. Pratt, Theobald Smith, E. E. Tyzzer, S. Burt Wolbach, Robert M. Yerkes.

Johns Hopkins University.—John J. Abel, Rufus I. Cole, Harvey W. Cushing, Percy M. Dawson, W. W. Ford, William H. Howell, Walter Jones, A. S. Loevenhart, W. G. MacCallum, F. P. Mall, William H. Welch.

Massachusetts Institute of Technology.—Percy G. Stiles.

McGill University (Montreal).—J. George Adami, Charles W. Duval, Oskar Klotz.

U. S. Public Health and Marine Hospital Service.—Walter R. Brinckerhoff (*Honolulu*), Reid Hunt (*Washington*), J. H. Kastle (*Washington*), M. J. Rosenau (*Washington*), Richard P. Strong (*Manila*).

University College (London).—Arthur R. Cushny.

University of Alabama.—William Salant.

University of Buffalo.—G. H. A. Clowes, Herbert U. Williams.

University of California.—Jacques Loeb, T. Brailsford Robertson, Alonzo E. Taylor.

University of Chicago.—R. R. Bensley, A. J. Carlson, Ludvig Hektoen, Edwin O. Jordan, Waldemar Koch, Albert P. Mathews, H. T. Ricketts, H. Gideon Wells.

University of Illinois.—Philip B. Hawk, Ward J. MacNeal.

University of Japan.—Naohidé Yatsu.

University of Michigan.—Carl G. Huber, Warren P. Lombard, Frederick G. Novy, Victor C. Vaughan, Aldred S. Warthin.

University of Missouri.—Robert B. Gibson.

University of Pennsylvania.—Alexander C. Abbott, E. G. Conklin, David L. Edsall, Ralph S. Lillie, Leo Loeb, Edward T. Reichert, J. Edwin Sweet.

University of Southern California (Los Angeles).—Lyman B. Stookey.

University of Syracuse.—Gustave M. Meyer.

University of Toronto.—A. B. Macallum.

University of Virginia.—C. H. Bunting.

University of Wisconsin.—Charles R. Bardeen, Joseph Erlanger.

Western Reserve University (Cleveland).—George W. Crile, J. J. R. Macleod, Torald Sollmann, G. N. Stewart.

Wistar Institute of Anatomy (Philadelphia).—H. H. Donaldson, Shinkishi Hatai.

Yale University.—R. H. Chittenden, Ross G. Harrison, Yandell Henderson, Lafayette B. Mendel, Frank P. Underhill, L. L. Woodruff.

Members present at the twenty sixth meeting:

Adler, Auer, Berg, Burton-Opitz, Brooks, Carrel, Crampton (C. W.), Ewing, Field, Flexner, Gies, Lee, Levene, Levin, Loeb (L.), Meltzer, Murlin, Norris, Salant, Terry, Weil.

Members elected at the twenty sixth meeting:

Edwin J. Banzhaf, William N. Berg, H. D. Dakin, Charles A. Elsberg, Shinkishi Hatai, Charles E. Simon, S. Burt Wolbach.

Dates of the next two regular meetings:

February 19, 1908 (fifth annual business meeting).

April 15, 1908.

PRESS OF
THE NEW ERA PRINTING COMPANY
LANCASTER, PA.

CLASSIFIED LIST OF MEMBERS OF THE SOCIETY FOR EXPERIMENTAL BIOLOGY AND MEDICINE.

Resident (Greater New York).

Bellevue Hospital.—Thomas Flournoy, Charles Norris.

Columbia University.—Russell Burton-Opitz, Gary N. Calkins, Henry E. Crampton, Richard H. Cunningham, Haven Emerson, Nellis B. Foster, William J. Gies, Christian A. Herter, Philip H. Hiss, Frederic S. Lee, T. H. Morgan, Horst Oertel, Alfred N. Richards, H. C. Sherman, Augustus B. Wadsworth, Edmund B. Wilson, Francis C. Wood.

Cornell University Medical College.—S. P. Beebe, B. H. Buxton, William J. Elser, James Ewing, Robert A. Hatcher, Philip A. Shaffer, Oscar Teague, John C. Torrey, Richard Weil, C. G. L. Wolf.

New York Department of Education.—C. Ward Crampton.

New York Department of Health.—James P. Atkinson, Cyrus W. Field.

New York Hospital.—Douglas Symmers.

New York Polyclinic Medical School.—Isaac Adler.

New York University.—Harlow Brooks, Edward K. Dunham, Graham Lusk, Arthur R. Mandel, John A. Mandel, John R. Murlin, William H. Park, George B. Wallace.

Rockefeller Institute for Medical Research.—John Auer, Alexis Carrel, Simon Flexner, James W. Jobling, P. A. Levene, S. J. Meltzer, Hideyo Noguchi, Eugene L. Opie, B. T. Terry.

St. Francis Hospital.—Fritz Schwyzer.

Sydenham Hospital.—Isaac Levin.

---

19 W. 42d Street.—Isaac F. Harris.

43 W. 52d Street.—Ludwig Kast.

Non-Resident.

Albany Medical College.—Holmes C. Jackson, Richard M. Pearce.

Buffalo University.—G. H. A. Clowes, Herbert U. Williams.

Carnegie Institution of Washington.—Francis G. Benedict (*Nutrition Laboratory, Boston*), Charles B. Davenport (*Station for Experimental Evolution, Cold Spring Harbor, N. Y.*), D. T. MacDougal (*Washington*).

Chicago University.—R. R. Bensley, A. J. Carlson, Ludvig Hektoen, Edwin O. Jordan, Waldemar Koch, Albert P. Mathews, H. T. Ricketts, H. Gideon Wells.

Connecticut Agricultural Experiment Station (New Haven).—Thomas B. Osborne.

Cooper Medical College (San Francisco).—William Ophüls.

Harvard University.—Carl L. Alsberg, Walter B. Cannon, W. T. Councilman, Otto Folin, Frederick P. Gay, Paul A. Lewis, G. H. Parker, Franz Pfaff, W. T. Porter, Joseph H. Pratt, Theobald Smith, E. E. Tyzzer, Robert M. Yerkes.

Johns Hopkins University.—John J. Abel, Rufus I. Cole, Harvey W. Cushing, Percy M. Dawson, W. W. Ford, William H. Howell, Walter Jones, A. S. Loevenhart, W. G. MacCallum, F. P. Mall, William H. Welch.

Massachusetts Institute of Technology.—Percy G. Stiles.

McGill University (Montreal).—J. George Adami, Charles W. Duval, Oskar Klotz.

Syracuse University.—Gustave M. Meyer.

U. S. Public Health and Marine Hospital Service.—Walter R. Brinckerhoff (*Honolulu*), Reid Hunt (*Washington*), J. H. Kastle (*Washington*), M. J. Rosenau (*Washington*), Richard P. Strong (*Manila*).

University College (London).—Arthur R. Cushny.

University of Alabama.—William Salant.

University of California.—Jacques Loeb, T. Brailsford Robertson, Alonzo E. Taylor.

University of Illinois.—Philip B. Hawk, Ward J. MacNeal.

University of Japan.—Naohidé Yatsu.

University of Michigan.—Carl G. Huber, Warren P. Lombard, Frederick G. Novy, Victor C. Vaughan, Aldred S. Warthin.

University of Missouri.—Robert B. Gibson.

University of Pennsylvania.—Alexander C. Abbott, E. G. Conklin, David L. Edsall, Ralph S. Lillie, Leo Loeb, Edward T. Reichert, J. Edwin Sweet.

University of Southern California (Los Angeles).—Lyman B. Stookey.

University of Toronto.—A. B. Macallum.

University of Virginia.—C. H. Bunting.

University of Wisconsin.—Charles R. Bardeen, Joseph Erlanger.

Western Reserve University (Cleveland).—George W. Crile, J. J. R. Macleod, Torald Sollmann, G. N. Stewart.

Wistar Institute of Anatomy (Philadelphia).—H. H. Donaldson.

Yale University.—R. H. Chittenden, Ross G. Harrison, Yandell Henderson, Lafayette B. Mendel, Frank P. Underhill, L. L. Woodruff.

Members present at the twenty fifth meeting:

Adler, Auer, Brooks, Burton-Opitz, Carrel, Crampton (C. W.), Emerson, Ewing, Field, Flexner, Foster, Gay, Gies, Hatcher, Jobling, Kast, Lee, Levene, Levin, Lewis, Lusk, Mandel (A. R.), Mandel (J. A.), Meltzer, Morgan, Murlin, Noguchi, Oertel, Park, Richards, Terry, Teague, Tyzzer, Wallace, Weil, Wolf, Wood.

Members elected at the twenty fifth meeting:

Edward T. Reichert, M. J. Rosenau, Richard P. Strong.

Dat s of the next two regular meetings:

December 18, 1907.

February 19, 1908 (fifth annual business meeting).

PRESS OF
THE NEW ERA PRINTING COMPANY
LANCASTER, PA.

PROCEEDINGS

OF THE

SOCIETY FOR

EXPERIMENTAL BIOLOGY AND MEDICINE

TWENTY EIGHTH MEETING

PHYSIOLOGICAL LABORATORY OF THE NEW YORK UNIVERSITY AND BELLEVUE HOSPITAL MEDICAL COLLEGE

NEW YORK CITY

APRIL 15, 1908

VOLUME V

No. 4

NEW YORK

MAY 15, 1908

CONTENTS.

GRAHAM LUSK : Influence of cold and exercise in phlorhizin glycosuria. 39 (295).

J. R. MURLIN : The influence of carbohydrate on the protein metabolism of a fasting pregnant dog. 40 (296).

W. S. HALSTED : The transplantation of parathyroid glands in dogs. 41 (297).

MARIE IMCHANITZKY : The nervous coördination of the auricles and ventricle of the heart of the lizard. (Communicated by S. J. MELTZER.) 42 (298).

LAFAYETTE B. MENDEL : The influence of diet on the chemical composition of the body. 43 (299).

LAFAYETTE B. MENDEL and TADASU SAIKI : The chemical composition of nonstriated mammalian muscle. 44 (300).

LORANDE LOSS WOODRUFF : Increased susceptibility of protozoa to poison due to treatment with alcohol. 45 (301).

F. P. GAY and E. E. SOUTHARD : The relative specificity of anaphylaxis. 46 (302).

W. G. MACCALLUM and CARL VOEGTLIN : On the relation of calcium metabolism to tetany and the cure of tetany by administration of calcium. 47 (303).

DON R. JOSEPH and S. J. MELTZER : The relative toxicity of the chlorides of magnesium, calcium, potassium and sodium. 48 (304).

JOHN AUER and S. J. MELTZER : The action of calcium upon the pupil and its relation to the effects of mydriatics. 49 (305).

ROBERT A. HATCHER and HAROLD C. BAILEY : The destruction of strophanthin in the animal organism. 50 (306).

J. J. R. MACLEOD : On the nature of the so-called glycogenolytic fibers in the greater splanchnic nerves. 51 (307).

SIMON FLEXNER : Prevention of syphilis in Macacus Rhesus by atoxyl. 52 (308).

SIMON FLEXNER and J. W. JOBLING : Further notes on a rat tumor. 53 (309).

JOHN A. MANDEL, W. A. JACOBS and P. A. LEVENE : On nucleic acids. 54 (310).

R. BURTON-OPITZ and DANIEL R. LUCAS : Regarding the innervation of the blood vessels of the kidney. 55 (311).

R. BURTON-OPITZ : Regarding the innervation of the blood vessels of the intestine. 56 (312).

PAUL A. LEWIS : Note on anaphylaxis to horse serum. 57 (313).

L. J. COHEN and WILLIAM J. GIES : A study of "protagon" prepared by the Wilson-Cramer method. 58 (314).

The proceedings of the Society for Experimental Biology and Medicine are published as soon as possible after each meeting. Regular meetings of the Society are held in New York on the third Wednesdays of October, December, February, April and May. A volume of the proceedings consists of the numbers issued during an academic year.

The price per volume, sent postage prepaid, is one dollar. The price of copies of the proceedings of any meeting is twenty cents each, postage prepaid. Subscriptions are payable in advance.

Address communications to any of the following members of the Council of the Society :

PAST PRESIDENTS — S. J. Meltzer, Rockefeller Institute for Medical Research ; Edmund B. Wilson, Columbia University ; Simon Flexner, Rockefeller Institute for Medical Research.

PRESIDENT — Frederic S. Lee, Columbia University.

VICE-PRESIDENT — Thomas H. Morgan, Columbia University.

TREASURER — Graham Lusk, New York University.

SECRETARY — William J. Gies, Columbia University.

MANAGING EDITOR — The Secretary, College of Physicians and Surgeons, 437 W. 59th St., New York City.

PROCEEDINGS

OF THE

SOCIETY FOR

EXPERIMENTAL BIOLOGY AND MEDICINE

TWENTY NINTH MEETING

CORNELL UNIVERSITY MEDICAL COLLEGE

NEW YORK CITY

MAY 20, 1908

VOLUME V

No. 5

(WITH TITLE PAGE, INDEX, ETC., FOR VOLUME V.)

NEW YORK

JULY 1, 1908

C. B. Davenport: Heredity of some human physical characteristics. 59 (315).

Leo Loeb: The experimental production of the maternal part of the placenta in the rabbit. 60 (316).

Elizabeth Cooke and Leo Loeb: Hemolytic action of the venom of Heloderma suspectum. 61 (317).

Thomas B. Osborne: The biological relations of seed proteins. 62 (318).

Nellis B. Foster and Adrian V. S. Lambert: Variation in hydrochloric acid secretion during the digestive period. 63 (319).

Nellis B. Foster and Adrian V. S. Lambert: The effects of some organic acids on the secretion of gastric juice. 64 (320).

Nellis B. Foster and Adrian V. S. Lambert: The effect of mechanical obstruction of the pyloric outlet on gastric secretion. 65 (321).

Isaac Levin and John H. Larkin: Transplantation of devitalized arterial segments. 66 (322).

Theodore C. Janeway and Herman O. Mosenthal: A study of nitrogen metabolism in a case presenting short paroxysms of fever of unknown origin. 67 (323).

Wilbur Ward: Histological changes in transplanted blood vessels. (Communicated by Francis Carter Wood.) 68 (324).

A. S. Loevenhart and D. R. Hooker: Note upon the supposed presence of a gastric hormon in the salivary glands. 69 (325).

Don R. Joseph: The relation of the weight of the stomach- and cecum-contents to the body weight in rabbits. 70 (326).

Don R. Joseph: The inhibitory influence of magnesium upon some of the toxic effects of eserin. 71 (327).

L. B. Stookey: Influence of iodides on autolysis. 72 (328).

L. B. Stookey and Vera Gardner: Relation of the thyroids to autolysis. 73 (329).

L. B. Stookey: On the physiology of the thyroids. 74 (330).

L. B. Stookey and Vera Gardner: On the pharmacology of the iodides. 75 (331).

L. B. Stookey and A. Halden Jones: Glycogen formation from arabinose in chicks. 76 (332).

L. B. Stookey and Ethel L. Leonard: Is oxalic acid a product of hepatic uricolysis in man? 77 (333).

Lorande Loss Woodruff: The life cycle of paramecium. 78 (334).

Emily C. Seaman and William J. Gies: An examination of Bardach's new protein test. 79 (335).

William Weinberger (by invitation): A study of metabolic effects of experimental polycythemia in dogs. 80 (336).

Matthew Steel (by invitation): On the metabolic influence of magnesium sulfate in dogs, with special reference to the partition of the nitrogenous constituents of the urine. 81 (337).

Matthew Steel and William J. Gies: On the determination of ammonia, by the Folin method, in urines containing crystalline ammonio-magnesium phosphate. 82 (338).

The proceedings of the Society for Experimental Biology and Medicine are published as soon as possible after each meeting. Regular meetings of the Society are held in New York on the third Wednesdays of October, December, February, April and May. A volume of the proceedings consists of the numbers issued during an academic year.

The price per volume, sent postage prepaid, is one dollar. The price of copies of the proceedings of any meeting is twenty cents each, postage prepaid. Subscriptions are payable in advance.

Address communications to any of the following members of the Council of the Society:

Past Presidents — S. J. Meltzer, Rockefeller Institute for Medical Research; Edmund B. Wilson, Columbia University; Simon Flexner, Rockefeller Institute for Medical Research.

President — Frederic S. Lee, Columbia University.

Vice-President — Thomas H. Morgan, Columbia University.

Treasurer — Graham Lusk, New York University.

Secretary — William J. Gies, Columbia University.

Managing Editor — The Secretary, College of Physicians and Surgeons,

PROCEEDINGS

OF THE

SOCIETY FOR

EXPERIMENTAL BIOLOGY AND MEDICINE

VOLUME V

1907–1908

EDITED BY THE SECRETARY

NEW YORK

JULY 1, 1908

C. B. Davenport: Heredity of some human physical characteristics. 59 (315).

Leo Loeb: The experimental production of the maternal part of the placenta in the rabbit. 60 (316).

Elizabeth Cooke and Leo Loeb: Hemolytic action of the venom of Heloderma suspectum. 61 (317).

Thomas B. Osborne: The biological relations of seed proteins. 62 (318).

Nellis B. Foster and Adrian V. S. Lambert: Variation in hydrochloric acid secretion during the digestive period. 63 (319).

Nellis B. Foster and Adrian V. S. Lambert: The effects of some organic acids on the secretion of gastric juice. 64 (320).

Nellis B. Foster and Adrian V. S. Lambert: The effect of mechanical obstruction of the pyloric outlet on gastric secretion. 65 (321).

Isaac Levin and John H. Larkin: Transplantation of devitalized arterial segments. 66 (322).

Theodore C. Janeway and Herman O. Mosenthal: A study of nitrogen metabolism in a case presenting short paroxysms of fever of unknown origin. 67 (323).

Wilbur Ward: Histological changes in transplanted blood vessels. (Communicated by Francis Carter Wood.) 68 (324).

A. S. Loevenhart and D. R. Hooker: Note upon the supposed presence of a gastric hormon in the salivary glands. 69 (325).

Don R. Joseph: The relation of the weight of the stomach- and cecum-contents to the body weight in rabbits. 70 (326).

Don R. Joseph: The inhibitory influence of magnesium upon some of the toxic effects of eserin. 71 (327).

L. B. Stookey: Influence of iodides on autolysis. 72 (328).

L. B. Stookey and Vera Gardner: Relation of the thyroids to autolysis. 73 (329).

L. B. Stookey: On the physiology of the thyroids. 74 (330).

L. B. Stookey and Vera Gardner: On the pharmacology of the iodides. 75 (331).

L. B. Stookey and A. Halden Jones: Glycogen formation from arabinose in chicks. 76 (332).

L. B. Stookey and Ethel L. Leonard: Is oxalic acid a product of hepatic uricolysis in man? 77 (333).

Lorande Loss Woodruff: The life cycle of paramecium. 78 (334).

Emily C. Seaman and William J. Gies: An examination of Bardach's new protein test. 79 (335).

William Weinberger (by invitation): A study of metabolic effects of experimental polycythemia in dogs. 80 (336).

Matthew Steel (by invitation): On the metabolic influence of magnesium sulfate in dogs, with special reference to the partition of the nitrogenous constituents of the urine. 81 (337).

Matthew Steel and William J. Gies: On the determination of ammonia, by the Folin method, in urines containing crystalline ammonio-magnesium phosphate. 82 (338).

The proceedings of the Society for Experimental Biology and Medicine are published as soon as possible after each meeting. Regular meetings of the Society are held in New York on the third Wednesdays of October, December, February, April and May. A volume of the proceedings consists of the numbers issued during an academic year.

The price per volume, sent postage prepaid, is one dollar. The price of copies of the proceedings of any meeting is twenty cents each, postage prepaid. Subscriptions are payable in advance.

Address communications to any of the following members of the Council of the Society:

Past Presidents — S. J. Meltzer, Rockefeller Institute for Medical Research; Edmund B. Wilson, Columbia University; Simon Flexner, Rockefeller Institute for Medical Research.

President — Frederic S. Lee, Columbia University.

Vice-President — Thomas H. Morgan, Columbia University.

Treasurer — Graham Lusk, New York University.

Secretary — William J. Gies, Columbia University.

Managing Editor — The Secretary, College of Physicians and Surgeons, [illegible] St., New York City.

CLASSIFIED LIST OF MEMBERS OF THE SOCIETY FOR EXPERIMENTAL BIOLOGY AND MEDICINE.

Resident (Greater New York).

Bellevue Hospital.—Thomas Flournoy, Charles Norris, Alwin M. Pappenheimer.

Columbia University.—Russell Burton-Opitz, Gary N. Calkins, Henry E. Crampton, Richard H. Cunningham, Haven Emerson, Nellis B. Foster, William J. Gies, Christian A. Herter, Philip H. Hiss, Frederic S. Lee, Isaac Levin, Gustave M. Meyer, Thomas H. Morgan, Henry C. Sherman, Augustus B. Wadsworth, Edmund B. Wilson, Francis C. Wood.

Cornell University Medical College.—S. P. Beebe, B. H. Buxton, William J. Elser, James Ewing, Robert A. Hatcher, Philip A. Shaffer, John C. Torrey, Richard Weil, C. G. L. Wolf.

Mt. Sinai Hospital.—Charles A. Elsberg.

New York Department of Education.—C. Ward Crampton.

New York Department of Health.—James P. Atkinson, Edwin J. Banzhaf, L. W. Famulener, Cyrus W. Field.

New York Hospital.—Douglas Symmers.

New York Polyclinic Medical School.—Isaac Adler.

New York Post-Graduate Medical School.—Ludwig Kast.

New York University.—Harlow Brooks, Edward K. Dunham, Graham Lusk, Arthur R. Mandel, John A. Mandel, John R. Murlin, William H. Park, Richard M. Pearce, George B. Wallace.

Rockefeller Institute for Medical Research.—John Auer, Alexis Carrel, Simon Flexner, Walter A. Jacobs, James W. Jobling, Don R. Joseph, P. A. Levene, S. J. Meltzer, Hideyo Noguchi, Eugene L. Opie, B. T. Terry, Donald D. Van Slyke.

Sage Institute of Pathology, City Hospital.—Theodore C. Janeway, Horst Oertel.

St. Francis Hospital.—Fritz Schwyzer.

819 Madison Avenue.—H. D. Dakin.

449 E. 57th Street.—Isaac F. Harris.

Non-Resident.

Albany Medical College.—Holmes C. Jackson.

Baltimore Medical College.—Charles E. Simon.

Carnegie Institution of Washington.—Francis G. Benedict (*Nutrition Laboratory, Boston*), Charles B. Davenport (*Station for Experimental Evolution, Cold Spring Harbor, N. Y.*), D. T. MacDougal (*Washington*), Alfred G. Mayer (*Marine Laboratory, Tortugas, Fla.*).

Connecticut Agricultural Experiment Station (New Haven).—Thomas B. Osborne.

Cooper Medical College (San Francisco).—William Ophüls.

Harvard University.—Carl L. Alsberg, Walter B. Cannon, W. T. Councilman, Otto Folin, Frederick P. Gay, Paul A. Lewis, G. H. Parker, Franz Pfaff, W. T. Porter, Joseph H. Pratt, Theobald Smith, E. E. Tyzzer, S. Burt Wolbach, Robert M. Yerkes.

Johns Hopkins University.—John J. Abel, Rufus I. Cole, Harvey W. Cushing, W. W. Ford, W. S. Halsted, William H. Howell, Walter Jones, W. G. MacCallum, F. P. Mall, William H. Welch.

Massachusetts Institute of Technology.—Percy G. Stiles.

McGill University (Montreal).—J. George Adami, Charles W. Duval, Oskar Klotz.

Medico-Chirurgical College (Philadelphia).—Isaac Ott.

Northwestern University Medical School (Chicago).—J. B. Murphy, Alfred N. Richards.

Princeton University.—Edwin G. Conklin.

U. S. Department of Agriculture (Washington, D. C.).—Wm. N. Berg, William Salant.

U. S. Department of the Interior, Bureau of Science, Manila.—Oscar Teague.

U. S. Public Health and Marine Hospital Service.—Walter R. Brinckerhoff (*Honolulu*), Reid Hunt (*Washington*), J. H. Kastle (*Washington*), M. J. Rosenau (*Washington*), Richard P. Strong (*Manila*).

University College (London).—Arthur R. Cushny.

University of Buffalo.—G. H. A. Clowes, Herbert U. Williams.

University of California.—Jacques Loeb, T. Brailsford Robertson, Alonzo E. Taylor.

University of Chicago.—R. R. Bensley, A. J. Carlson, Ludvig Hektoen, Edwin O. Jordan, Waldemar Koch, Frank R. Lillie, Albert P. Mathews, H. T. Ricketts, H. Gideon Wells.

University of Illinois.—Philip B. Hawk, Ward J. MacNeal.

University of Japan.—Naohidé Yatsu.

University of Michigan.—Otto C. Glaser, Carl G. Huber, Warren P. Lombard, Frederick G. Novy, Victor C. Vaughan, Aldred S. Warthin.

University of Missouri.—C. Stuart Gager, Robert B. Gibson.

University of Pennsylvania.—Alexander C. Abbott, David L. Edsall, Ralph S. Lillie, Leo Loeb, Edward T. Reichert, J. Edwin Sweet.

University of Southern California (Los Angeles).—Lyman B. Stookey.

University of Toronto.—A. B. Macallum.

University of Virginia.—C. H. Bunting.

University of Wisconsin.—Charles R. Bardeen, Joseph Erlanger, Arthur S. Loevenhart.

Western Reserve University (Cleveland).—George W. Crile, J. J. R. Macleod, Torald Sollmann, G. N. Stewart.

Wistar Institute of Anatomy (Philadelphia).—H. H. Donaldson, Shinkishi Hatai.

Yale University.—R. H. Chittenden, Ross G. Harrison, Yandell Henderson, Lafayette B. Mendel, Frank P. Underhill, L. L. Woodruff.

Members present at the twenty ninth meeting:

Auer, Beebe, Carrel, Davenport, Elser, Ewing, Famulener, Foster, Gies, Harris, Hatcher, Janeway, Joseph, Kast, Levene, Levin, Loeb (L.), Meltzer, Murlin, Noguchi, Osborne, Salant, Wadsworth, Wallace, Wood.

Dates of the next two regular meetings:

October 21, 1908. December 16, 1908.

By mistake the title page of Volume V of the Proceedings of the Society for Experimental Biology and Medicine was printed also on the cover of No. 5 of that volume. The first two pages of this folder are the correct front cover of the last number of the aforesaid Proceedings and may be used as indicated in the second paragraph of the preface of Volume V.

CLASSIFIED LIST OF MEMBERS OF THE SOCIETY FOR EXPERIMENTAL BIOLOGY AND MEDICINE.

Resident (Greater New York).

Bellevue Hospital.—Thomas Flournoy, Charles Norris, Alwin M. Pappenheimer.

Columbia University.—Russell Burton-Opitz, Gary N. Calkins, Henry E. Crampton, Richard H. Cunningham, Haven Emerson, Nellis B. Foster, William J. Gies, Christian A. Herter, Philip H. Hiss, Frederic S. Lee, Isaac Levin, T. H. Morgan, Alfred N. Richards, H. C. Sherman, Augustus B. Wadsworth, Edmund B. Wilson, Francis C. Wood.

Cornell University Medical College.—S. P. Beebe, B. H. Buxton, William J. Elser, James Ewing, Robert A. Hatcher, Philip A. Shaffer, John C. Torrey, Richard Weil, C. G. L. Wolf.

Mt. Sinai Hospital.—Charles A. Elsberg.

New York Botanical Garden.—C. Stuart Gager.

New York Department of Education.—C. Ward Crampton.

New York Department of Health.—James P. Atkinson, Edwin J. Banzhaf, L. W. Famulener, Cyrus W. Field.

New York Hospital.—Douglas Symmers.

New York Polyclinic Medical School.—Isaac Adler.

New York Post-Graduate Medical School.—Ludwig Kast.

New York University.—Harlow Brooks, Edward K. Dunham, Graham Lusk, Arthur R. Mandel, John A. Mandel, John R. Murlin, William H. Park, George B. Wallace.

Rockefeller Institute for Medical Research.—John Auer, Alexis Carrel, Simon Flexner, Walter A. Jacobs, James W. Jobling, Don R. Joseph, P. A. Levene, S. J. Meltzer, Hideyo Noguchi, Eugene L. Opie, B. T. Terry, Donald D. Van Slyke.

Sage Institute of Pathology, City Hospital.—Theodore C. Janeway, Horst Oertel.

St. Francis Hospital.—Fritz Schwyzer.

---

819 Madison Avenue.—H. D. Dakin.

449 E. 57th Street.—Isaac F. Harris.

Non-Resident.

Albany Medical College.—Holmes C. Jackson, Richard M. Pearce.

Baltimore Medical College.—Charles E. Simon.

Carnegie Institution of Washington.—Francis G. Benedict (*Nutrition Laboratory, Boston*), Charles B. Davenport (*Station for Experimental Evolution, Cold Spring Harbor, N. Y.*), D. T. MacDougal (*Washington*), Alfred G. Mayer (*Marine Laboratory, Tortugas, Fla.*).

Connecticut Agricultural Experiment Station (New Haven).—Thomas B. Osborne.

Cooper Medical College (San Francisco).—William Ophüls.

Harvard University.—Carl L. Alsberg, Walter B. Cannon, W. T. Councilman, Otto Folin, Frederick P. Gay, Paul A. Lewis, G. H. Parker, Franz Pfaff, W. T. Porter, Joseph H. Pratt, Theobald Smith, E. E. Tyzzer, S. Burt Wolbach, Robert M. Yerkes.

Johns Hopkins University.—John J. Abel, Rufus I. Cole, Harvey W. Cushing, W. W. Ford, W. S. Halsted, William H. Howell, Walter Jones, A. S. Loevenhart, W. G. MacCallum, F. P. Mall, William H. Welch.

Massachusetts Institute of Technology.—Percy G. Stiles.

McGill University (Montreal).—J. George Adami, Charles W. Duval, Oskar Klotz.

Medico-Chirurgical College (Philadelphia).—Isaac Ott.

Northwestern University Medical School (Chicago). — J. B. Murphy.

U. S. Department of Agriculture (Washington, D. C.).—Wm. N. Berg, William Salant.

U. S. Department of the Interior, Bureau of Science, Manila.—Oscar Teague.

U. S. Public Health and Marine Hospital Service.—Walter R. Brinckerhoff (*Honolulu*), Reid Hunt (*Washington*), J. H. Kastle (*Washington*), M. J. Rosenau (*Washington*), Richard P. Strong (*Manila*).

University College (London).—Arthur R. Cushny.

University of Buffalo.—G. H. A. Clowes, Herbert U. Williams.

University of California.—Jacques Loeb, T. Brailsford Robertson, Alonzo E. Taylor.

University of Chicago.—R. R. Bensley, A. J. Carlson, Ludvig Hektoen, Edwin O. Jordan, Waldemar Koch, Frank R. Lillie, Albert P. Mathews, H. T. Ricketts, H. Gideon Wells.

University of Illinois.—Philip B. Hawk, Ward J. MacNeal.

University of Japan.—Naohidé Yatsu.

University of Michigan.—Otto C. Glaser, Carl G. Huber, Warren P. Lombard, Frederick G. Novy, Victor C. Vaughan, Aldred S. Warthin.

University of Missouri.—Robert B. Gibson.

University of Pennsylvania.—Alexander C. Abbott, E. G. Conklin, David L. Edsall, Ralph S. Lillie, Leo Loeb, Edward T. Reichert, J. Edwin Sweet.

University of Southern California (Los Angeles).—Lyman B. Stookey.

University of Syracuse.—Gustave M. Meyer.

University of Toronto.—A. B. Macallum.

University of Virginia.—C. H. Bunting.

University of Wisconsin.—Charles R. Bardeen, Joseph Erlanger.

Western Reserve University (Cleveland).—George W. Crile, J. J. R. Macleod, Torald Sollmann, G. N. Stewart.

Wistar Institute of Anatomy (Philadelphia).—H. H. Donaldson, Shinkishi Hatai.

Yale University.—R. H. Chittenden, Ross G. Harrison, Yandell Henderson, Lafayette B. Mendel, Frank P. Underhill, L. L. Woodruff.

Members present at the twenty eighth meeting:

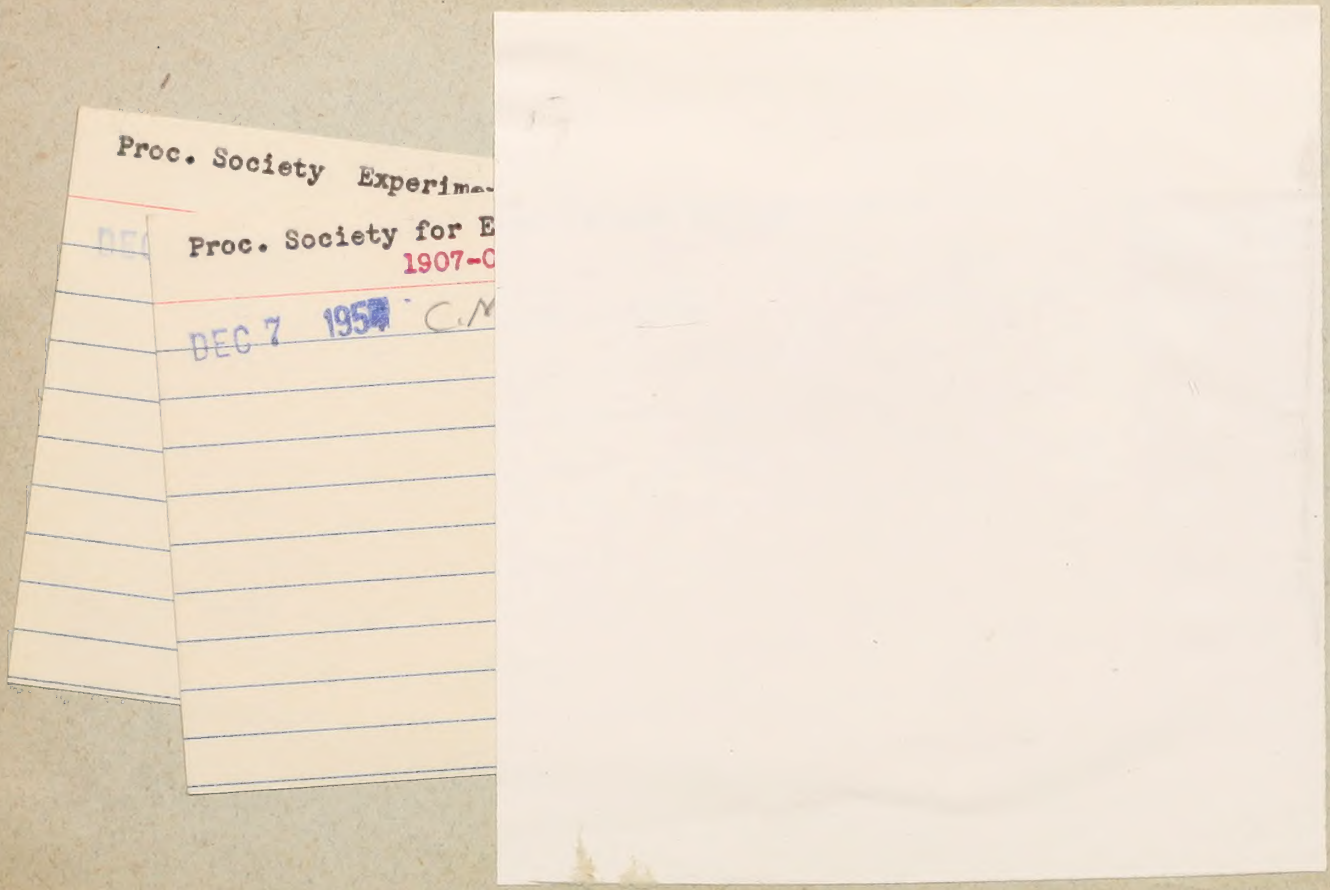
Auer, Carrel, Elsberg, Famulener, Flexner, Foster, Gay, Gies, Halsted, Hatcher, Jacobs, Jobling, Joseph, Lee, Levene, Lusk, MacCallum (W.G.), Mandel (A.R.), Mandel (J. A.), Meltzer, Mendel, Murlin, Noguchi, Shaffer, Teague, Terry, Torrey, Van Slyke, Wallace, Woodruff.

Members elected at the twenty eighth meeting:

Otto C. Glaser, Alfred G. Mayer, J. B. Murphy, Isaac Ott.

Date of the next regular meeting:

October 21, 1908.


Proc. Society Experim
Proc. Society for E
1907-C
DEC 7 195
C.M

AMNH LIBRARY
100209745